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## LIPIODOL AND THE LUNGS.[1]

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(From the Baker Institute, Melbourne.)

IT was only natural that Sicard,[(1)] satisfied with the results of lipiodol as a diagnostic agent in lesions of the central nervous system, should proceed to find wider fields for its use. So, some twelve months after the spinal lipiograms appeared, the bronchial tree was similarly illuminated. Some American contributors to this subject refer to Chevalier Jackson[(2)] as its pioneer, for in his very wonderful bronchoscopic work he used to drop some powdered bismuth on to his field of exploration and got thus a clearer conception of the lesion. But even some four years before that (1915) a very effective filling of the lung with a contrast medium had happened. I say "happened" because it was accidental. Beeler,[(3)] of New York, while doing a bismuth meal was astounded to see a perfect outline of the right bronchial tree unfold itself as he watched the œsophageal descent of bismuth—there was an unsuspected œsophageo-tracheal fistula. One would have expected some enterprising contemporary American quick to take advantage of this opening, but though Gottlieb followed up Beeler's accident by using a contrast medium in his work on the lungs of dogs, it was not till 1922, the very year in which Sicard and Forestier were perfecting their technique in Paris, that Lynah,[(4)] of New York, took advantage of the Beeler incident in filling the lung with bismuth and oil for routine examination. These were only tentative efforts, to be eclipsed by the successful launching of Sicard's method and medium. Now, there had always been a

[1] The second part of a paper read at a meeting of the Victorian Branch of the British Medical Association on April, 1931. The first part was published in the issue of August 22, 1931.

reluctance to direct lung medication. The honour of introducing such medication seems to be doubtfully shared by Horace Green, of London, and A. Rosenberg,[5] of Vienna, half a century ago.

The usual medium was menthol or iodoform in olive oil till Lukens[6] introduced chaulmoogra oil. They were constantly being bombarded by experimental pathologists till at last, in 1917, the extensive experiments on rabbits and guinea-pigs by Waters and Rowntree[7] showed that the beneficial effects of lung medication more than compensated for the negligible epithelial spoiling resulting from the medium. "It is hoped," their article concluded, "that perfecting of the method may ultimately permit its use in man." Indeed, Campbell[8] had already reported having injected as much as one hundred cubic centimetres of menthol-oil in twenty-four hours without any harm.

So Sicard and Forestier's bold move with lipiodol was certain to raise a storm of opposition, and all the more in that the procedure was diagnostic only, not therapeutic.

Scarcely had the lung lipiograms appeared than there followed an article by the experimental pathologists, Corper and Freed,[9] of Denver, that began thus:

> Too frequently the haste occasioned by apparent clinical necessity leads to the introduction of a diagnostic or therapeutic measure without full scientific knowledge of the detailed action of such procedures.

And there followed a rather damnatory report based on extensive experiments on rabbits and guinea-pigs.

Five years later (1927) an almost identical introduction marked an article by a noted surgeon, Archibald,[10] of Montreal:

> It is a matter of common observation that the advent of a new therapeutic or diagnostic agent may be accompanied by such a wave of enthusiasm in its favour that its field of acceptance and approval early becomes extended beyond the limits that are warranted by the knowledge of its potential advantages and drawbacks.

It might be convenient at this early stage to review the opposition to lipiodol by reason of its supposed dangers or disadvantages. But before doing so, I shall briefly recall to your mind the various methods of introducing lipiodol into the lung.

### Methods of Introduction.

The original route of Sicard and Forestier was by way of the cricothyreoid membrane; and tracheal puncture has remained the most effective method, and sometimes the only possible one, in adults. Refinements are constantly taking place, chiefly in needles; for example, Chandler's[11] straight trocar and cannula, which is now widely used. At the Alfred Hospital we have for some years used a curved trocar and cannula, somewhat like a miniature tracheotomy tube; and last year Armand-Delille,[12] of Paris, introduced a new form of trocar and cannula—almost a facsimile of our own.

It is rather amusing that a correspondence ran for a few weeks in *The British Medical Journal* as to whether a skin puncture with a knife should first be made. Anyone accustomed to use a trocar and cannula knows how perfectly fitting its parts must be to make an easy skin puncture, even in a resisting area like the chest. At all events, a small nick assures an easy introduction.

A word about technique.

A small intradermal wheal is raised just above the cricoid cartilage, the patient lying on his back with head extended, but not uncomfortably so. A small puncture is made in the wheal down to the trachea; the operator, in front and to the left of the patient, grips the cricoid with the left thumb and forefinger and fixes it, and the patient, being impressed not to swallow for a second or two, the trocar and cannula is passed backwards and downwards into the trachea. To the heft of the cannula are attached small "lugs" for the securing of tapes, to be tied firmly round the neck for the safe retention of the cannula in the trachea. The patient is turned at once on to the desired side, and one cubic centimetre of 5% cocaine slowly run in. He is then taken to the X ray room and lipiodol is introduced, the patient lying on the side required. A six-inch length of small rubber tubing connecting the cannula with the lipiodol syringe makes the introduction of the warmed lipiodol easier. We have never found it necessary to use a syringe with a screw piston action. Another cubic centimetre of cocaine may be required in the course of the procedure.

No preparation is required, except a good cough linctus before and sometimes one during the procedure. The routine testing of the patient's tolerance for iodide with potassium iodide for a couple of days is practised in some clinics.

The tracheal method has its pitfalls and mishaps. A false passage may be made and the lipiodol may be delivered into the tissues of the neck; considerable emphysema of neck and face may result from excessive coughing; a pustule may form at the seat of puncture. We have had experience of all these with no bad after-effects. Forestier himself had a case of *œdema glottidis*, and Boidin[13] acute œdema of the larynx, both of which quickly subsided, and Lorey[14] reported three cases of disturbing cocainism, while in one of Olmer's[15] cases a severe epileptic seizure followed the tracheal puncture, and Leroux and Bouchet[16] report a fatal phlegmon of the neck.

There remain three other methods to consider.

*Bronchoscopic Method.*—We are quite content to leave the bronchoscopic method in the hands of the many budding Chevalier Jacksons in our midst.

*Transglottal Method.*—The transglottal method consists in the production of local (or general) anæsthesia and the passing of an introducer, such as a metal tipped catheter, into the trachea by the mouth, or by the nose (Haslinger's method).

*Supraglottal Method.*—In regard to the supraglottal method, Nather[17] found that the patient with an anæsthetized pharynx and glottis swallowed the "wrong way." Lipiodol introduced on to the back of the tongue entered, on the patient's being told to swallow, not the pharynx, but the larynx. Ochsner's[18] "passive" introduction was a somewhat similar method.

The latest of all is Singer's,[19] of Baltimore.

With the patient sitting and leaning towards the desired side, the nozzle of the lipiodol syringe is placed between

the uvula and the posterior pillar of the tonsillar space and the patient is told not to swallow, but to inhale rather deeply. The lipiodol introduced thus finds its way into the trachea, delivery being made only during inspiration; all this in an unanæsthetized throat.

This promises much in the way of speed and comfort. Personally, I have never been able to get anything but a scanty amount of lipiodol introduced. This method is successfully used at Saint Vincent's Hospital by Dr. Haydon.

Dr. Middleton has treated scores of children at the Children's Hospital by the transglottal method with a catheter, under a general anæsthetic—the ideal way for children. This is the method of Armand-Delille[20] with children. He, however, uses local anæsthesia.

Of the transglottal introducers there are many varieties, the outstanding ones being Iglauer's,[21] Myerson's[22] and Baisch's[23]—all more or less modified intubation apparatus.

### Dangers and Disadvantages.

#### *Iodism.*

The rarity of iodism may at first seem strange. It appears settled, however, that this very stable combination of iodine is irreducible in the lung; and whatever absorption of iodine there is must be from the overflow into the stomach, the acid contents of which apparently can dissociate the iodine; yet we have often had ten to twenty cubic centimetres in the stomach without any ill effect.

In spite of the very interesting articles of Roger and Binet[24] on the "Internal Digestion of Lung Surfaces," Knipping and Poundorf[25] showed in a series of lipiodol injections into the lungs of dogs that no iodine was found in the urine, unless a tracheo-œsophageal fistula were present.

Only one death is recorded in all the literature, although one case of disturbing iodine intoxication is reported by Rist[26] in the Paris Hospital records, a similar one by Lilbert[27] and a third by Belote.[28] O'Donovan,[29] of the London Hospital, reported the death of a patient who suffered from gross (?) iodine eruptions a few weeks after injection. Curiously enough no iodine was ever demonstrable in the urine.

#### *Vital Capacity.*

With the vital pulmonary capacity standing at 5,000 cubic centimetres, the introduction of, say, forty cubic centimetres of lipiodol does not seem alarming, but in the light of recent work it appears that the vital capacity may become extraordinarily reduced in certain pathological conditions of the lung and heart, and that in those circumstances the effect of a comparatively small amount of lipiodol may have to be taken into consideration in this direction. At any rate, under this heading one might place the sudden death recorded by Pirie[30] after lipiodol. This, of course, must be extremely rare. Besides, Pirie's case was one of advanced bilateral tuberculosis.

#### *Local Reaction.*

So far as local reactions are concerned, the early depressing findings of experimental pathologists have given place to such reports as those of Bettman and Kelly,[31] who in a large series of lung lipiograms on dogs failed to find any pathological disturbance of pulmonary epithelium.

#### *Lipiodol and Tuberculosis.*

Then comes the question of lipiodol and tuberculosis. While this is a serious consideration, the early dictum that lipiodol should not be used in tuberculous cases has been either discounted or greatly modified.

Archibald,[32] two years before he wrote the warning already referred to, published a series of signal successes with lipiodol, which included many tuberculous cases. In the interim he had a rather tragic death from a rapid pneumonia following lipiodol injection by the transglottic method in a tuberculous patient whose lesions were advanced and bilateral.

Two other acute pneumonias in tuberculous patients are found in the literature, one from Haslinger's[33] clinic in Vienna and one reported by Lichtwitz,[34] of Berlin. It is conceivable that, as in these pneumonic complications the transglottal method was used, the question of the possible introduction of the infective organism in this way may have to be considered. Fleischner,[35] of Vienna, pronounced against its use in tuberculous subjects, and Armand-Delille and Moncrieff[36] are content to say, generally, that patients with frank tuberculosis should not be submitted to this method; and yet one of their signal successes was in a case pronounced tuberculous as a result of ordinary X ray examination and shown by lipiodol to be bronchiectasis.

On the other hand, Iglauer and Kuhn[37] do not hesitate to use it in tuberculous cases in spite of their joint pronouncement: "Experience indicates that iodised oil may at times activate tuberculous lesions." Pirani[38] has never seen any harm follow in tuberculous cases, and Burrell,[39] of Brompton, has used it extensively with no resulting mishap.

Nobody, of course, would be likely to use lipiodol in an acute case of tuberculosis with high temperature and gross toxic symptoms, but our experience has been that of most recent workers, namely, that lipiodol in selected cases of tuberculosis, so far from being harmful, may be therapeutically useful.

This comparatively small array of disasters is challenged by an ever-increasing output from clinics all over the world recording no mishaps. Probably the most striking is the series of six hundred cases from Pritchard, Whyte and Gordon,[40] of Philadelphia, published two years ago, without a single accident. The Ballon[41] brothers, of Denver, report one hundred cases with similar results, and other clinics too numerous to detail here report in similar terms.

And so, in spite of its unfavourable reception, chiefly in experimental pathology quarters, the clarity it added to the ofttimes obscure ordinary X ray films, together with notable absence of any mischievous effects, made its success assured, and lately the tables seem to have been completely turned on even the experimental pathologists, when its therapeutic value began to be emphasized.

At the close of his address to an Edinburgh audience in 1927 Forestier[42] said:

It is a great principle in medicine *primum non nocere*, and I believe when you use lipiodol, you may do it sometimes with success, sometimes with failure, but never with any harm to the patient.

That was five years ago, and we may not now be inclined to subscribe to that *in toto*. At the Alfred Hospital, at any rate, we can say that the intervening years have only added to its successes, while the possible dangers have remained insignificant.

### Diagnosis and Research.

Is there any need for lipiodol? I am sure that not even the most ardent and accomplished radiologist would be slow in admitting the limitations of his art. He has seen his vague terminology of "honeycombing," linear markings, irregular densities, heart displacements, replaced by the positive lipiogram findings in, for example, bronchiectasis. It may be the sole arbiter between bronchiectatic enlargement on the one hand and tuberculous cavities or pulmonary abscesses on the other; between upper and lower lung lesions. The lung can no longer conceal itself behind the heart. In both artificial pneumothorax and thoracoplasty the efficiency of collapse becomes clearer. Pressure of a neoplasm on a bronchus is revealed in a striking way. Its aid may even be invoked in clarifying conditions where there is an apparently causeless persistence of pulmonary symptoms. The reports of cases trenching on all these avenues of diagnosis are overwhelmingly favourable.

Hartung,[43] of Illinois, in reporting last year his large series of lung lesions, admits to only 60% of his bronchiectatic cases being detectable by ordinary X ray.

Hedblom,[44] of New York, in an equally extensive series, states frankly that bronchiectasis with much fibrosis is undemonstrable by ordinary X rays; that pulmonary abscess may be quite indistinguishable from surrounding pulmonitis or sclerosis, or masked by thick pleura; and the nature, extent and relation of fistulous openings may be unknown, and in conjunction with Head demonstrated its value in thoracic surgery, particularly to clear up disappointing results in some thoracoplasties.

Ballon,[45] of Denver, in his series of ninety-four pulmonary abscesses says that quite a large percentage were indeterminable by ordinary X rays.

*Per contra*, Emile Sergent[46] gives six cases, reported pulmonary abscess in the routine X rays procedure, that were negatived by lipiodol. Nicaud and Dollfus[47] report two cases regarded as tuberculous for two years, unmasked as bronchiectasis by lipiodol, and from the great mass of general and expected successes of lipiodol comes an ever increasing number of what may be called refinements of diagnosis. Bezancon, Weil, Bernard and Azoulay[48] pointed out that till the advent of lipiodol no convincing picture was ever made of what the French call "*sèche hémoploique*" (the dry hæmorrhagic) type of bronchiectasis. The diagnosis of interlobar pleurisy in children was never satisfactory till demonstrated by Armand-Delille by means of lipiodol.

W. E. Lloyd's[49] pictures made familiar to us the characteristic rat-tailed appearance of the lipiodol in a bronchus partially obstructed by a neoplasm, and others which helped to differentiate an infiltrating from a merely displacing growth. Hedblom[50] reported one such striking diagnosis by lipiodol.

Again, Faulkner and Brunn,[51] of San Francisco, in their recent work with lipiodol, introduced the term "internal drainage" in relating the history of several cases in which the contents of a cavity were transferred to a distant part of the same lung or to the other lung, calling attention, by added physical signs thus produced, to what seemed to be further mischief other than in the *fons et origo* of the whole condition. This condition they show could only have been elucidated by lipiodol.

Jacobaeus[52] is well known as the pioneer in cautery section of pleural adhesions in the course of air compression of lung. Tragedies in this direction were not unknown. He found that lipiodol would insinuate itself into any bit of lung incorporated in what seemed to be an adhesion only, and the operator was thereby saved possible disaster.

J. D. Lawson,[53] of New York, never fails to define the size and nature of empyema cavities by lipiodol.

Following on Reinberg's[54] work in this direction, a very striking advance in the use of lipiodol appeared quite recently under the names of Hudson and Jarre,[55] of Michigan, in an article entitled "Lung Peristalsis." By means of what they called a "cin-ex. movey camera," pulmonary movements that hitherto were only suspected, were quite clearly demonstrated by the use of lipiodol. As may well be imagined, this is fraught with much physiological interest and may have far-reaching clinical results.

All these successes have been appropriately rounded off by very important papers appearing recently in *La Presse Médicale*, of Paris, from Sicard's, the originator's, own clinic; one from the pens of Bonnamour and Badolle[56] on "Cadaveric Lung Lipiograms"; another, more important still, by Sergent and Cottenot,[57] entitled "Normal Lung Lipiograms."

This side of the work, of course, cannot be too strongly exploited, and no radiologist can afford to neglect the study of these recent lung lipiograms. Introducing lipiodol into the bronchial tree is one thing, its interpretation another and a much more difficult matter.

One might turn aside here to inquire of the nature of the reception of lipiodol in England.

Clive Rivière,[58] always conservative, has made only half-hearted use of it. Burrell and Melville,[59] of the Brompton Hospital, are enthusiastic users of it. Burrell acknowledged his indebtedness to it in one striking instance. He had asked Roberts (of the Brompton and Saint Bartholomew's Hospitals) to do a thoracoplasty on one side, quite convinced by the ordinary X ray of the favourable condition of the other lung. It occurred to him to make first a lipiodol injection on the supposed sound side, and a chain of quite marked bronchiectatic dilatations was disclosed and the thoracoplasty was abandoned. Roberts himself makes constant use of lipiodol in his extensive surgical lung practice.

Chandler and Young,[60] of Victoria Park, have published a long series of successes with lipiodol. The former, who pronounced the introduction of lipiodol to be the greatest advance in pulmonary diagnosis since X rays themselves were introduced, has to his credit, in collaboration with Burton Wood, a short but quite good book on lipiodol and the methods of its use, as well as a particular kind of trocar and cannula for tracheal puncture, now in general use. The latter, Burton Wood,[61] had the good fortune early to unmask a case of upper lobe bronchiectasis, the discovery of which by lipiodol made him a very ardent supporter of its routine use.

But its strongest outpost was to be in Glasgow. There, in the well known Children's Clinic, Findlay and Graham[62] explored it to the fullest. "It was with the greatest interest," say Findlay and Graham, "that we availed ourselves of the help of lipiodol and as a result of its use we have modified our opinion, that chronic pneumonia is always accompanied by bronchiectasis. From our premature confidence in the reliability of physical signs we were at first inclined to the view that the use of lipiodol would be chiefly academic, but we feel now the method to be essential for the accurate diagnosis of lesions and their extent."

In Edinburgh, Guy, Elder and Lloyd[63] have added their quota of successes to the literature of the subject.

### Treatment.

Naturally, in regard to treatment we are on less sure ground. Just as in the earlier days experimental pathologists warned us of the danger to lung tissue that failed to eventuate clinically, so the bacteriologists have insisted that lipiodol has no bactericidal properties, *in vitro*; and yet the same clinical experience leads one to doubt this, *in vivo*.

It has been shown by Lincoln Brown[64] that the removal of lipiodol is by phagocytosis. This in itself is suggestive of benefit, and indeed Fiessinger and Lemaire[65] use lipiodol as a routine in stubborn tuberculous pleural effusion to produce leucocytosis. Neuswanger[66] (1926) found streptococci growing plentifully in lipiodol media. This was confirmed a year later by Chandler and Gloyne.[67] Dévé's[68] experiments on hydatid cysts with lipiodol gave equally negative results. In spite of this Ochsner maintains that he has counted organisms after lipiodol and found them constantly diminished. Mattice[69] puts forward the intriguing suggestion that lipiodol puts a protective coat over the inflamed bronchi and in this way is of benefit. Ransohoff[70] reports six cases of empyema in which he used lipiodol as a diagnostic agent only, with five quite unexpected recoveries without any other treatment.

Castex[71] reports his successful use of lipiodol infiltration in tuberculous pericarditis. Möller and Magnus,[72] of Stockholm, and Abramovitch,[73] of Ukraine, use lipiodol routinely in the treatment of bronchiectasis.

Steihm,[74] of Vienna, considered that nineteen of his patients with bronchiectasis were to all intents and purposes cured by lipiodol. Eschbach,[75] of Paris, at the end of last year reports the cleaning up of two cases of gangrene of the lung with three injections of twenty cubic centimetres of lipiodol at eight days' interval, and Sato[76] is equally emphatic of its value in suppurating conditions generally.

We had one conspicuous case of large abscess of the lung in which compression failed, clear up completely after repeated lipiodol injection. In this case the perseverance with lipiodol was at the patient's own request. One of our cases illustrates what Ballon speaks of as the effect of lipiodol in clearing up cases of persistent symptoms apparently causeless.

A boy had been a regular attendant at the outdoor clinic with a persistent cough and a good deal of sputum. X ray examination was questionably indicative of bronchiectasis. Lipiodol injection revealed a perfectly normal lung, and the boy up till now (eighteen months later) has required no further treatment for cough.

I have met no one with any considerable experience in the use of lipiodol who has not been impressed by its general good effect in almost every kind of pulmonary lesion.

### The Medium.

No consideration of the subject would be complete without some reference to the medium itself, lipiodol.

Lipiodol is a French preparation of Lafay, 40% iodine in poppy oil. The ascending lipiodol, introduced four years ago, is a quarter of the strength of the descending.

Sicard quite accidentally happened on this substance from having observed its opacity to X rays in cases in which it had been injected along the sciatic nerve therapeutically, a routine treatment on the Continent of some twenty-five years' standing. As a matter of fact, lipiodol fell into some disgrace before its use in spinal work.

Béclère[77] said he could not report on the condition of a hip owing to the surrounding lipiodol obscuring his view. On another occasion the same thing happened with a shoulder. He records an operation for a stone in the kidney for which some lipiodol in the tissues of the flank was responsible, and a pulmonary abscess that was diagnosed from a plaque of lipiodol in the back.

Germany's corresponding substance is Merck's "Iodipin." England responded very late with Martindale's "Iodinol." Suspicions of the safety of an iodized oil, especially in cases of tuberculosis, led Dyroff[78] to use a brominized oil; He called it "Contrastol." Rad,[79] of Vienna, reports nineteen cases of tuberculosis in which "Contrastol" was used without harm. Iglauer and Kuhn,[80] of Cincinatti, report eighteen tuberculous cases with "Contrastol" without mishap. Suffice it to say we have used lipiodol in as many cases of tuberculosis with equally good results. Putnam[81] confirmed experimentally its innocuousness on rabbits. The same innocuousness had been shown similarly in the case of lipiodol.

Frazier,[82] of Philadelphia, and Glaser,[83] of Los Angeles, gave the name "Campiodol" to a preparation consisting of iodized rapeseed oil. This they found equally innocuous in cerebral and spinal cord work. Frazier quite recently reported five recoveries from empyema, for which he had used "Campiodol" for diagnostic purposes only.

Schelenz,[84] of Berlin, uses iodized sesame oil with equal success and safety.

Blance,[85] of Indianapolis, forced to contrive something in the temporary lack of lipiodol supplies, made use of what he called a "home-made iodized corn oil," and had quite satisfactory and safe results.

### Cases.

In the supplement to this issue will be found a series of figures which, with their attached legends, will illustrate the paper sufficiently without detailed reports of cases.

### Acknowledgement.

Neither this article nor that on the use of lipiodol in the central nervous system would have been possible without the enthusiastic help and painstaking diagnosis of Dr. Love, the Alfred Hospital Radiologist.

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## ILLUSTRATIONS TO THE ARTICLE BY DR. J. F. MACKEDDIE.

FIGURE I.
Lateral view of a suspected bronchiectatic base. A normal lipiodol filling.

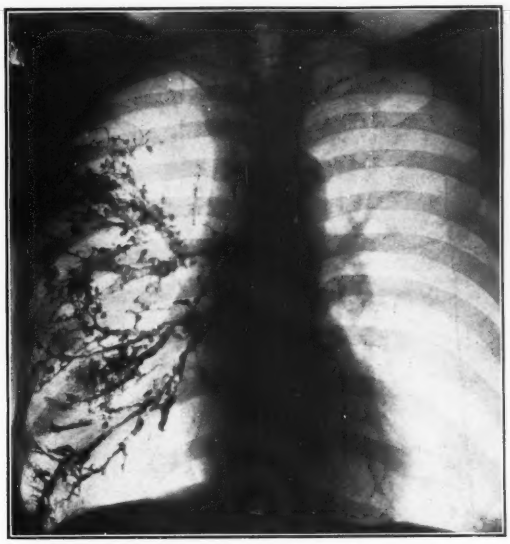

FIGURE II.
A suspected bronchial dilatation: a normal filling with abatement of symptoms after lipiodol.

FIGURE III.
Suspicion of right lung mischief dispelled by lipiodol.

FIGURE IV.
Appears at first sight to be an apical early bronchiectasis, but is a tuberculous lesion. This lesion was successfully compressed by air.

## ILLUSTRATIONS TO THE ARTICLE BY DR. J. F. MACKEDDIE.

FIGURE V.
Lateral view of general bronchial dilatation.

FIGURE VI.
A good lateral view of advancing unilateral cylindrical bronchiectasis.

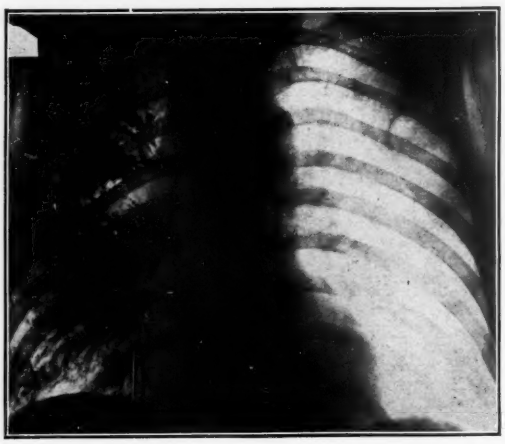

FIGURE VII.
Definite tuberculous lesion where signs were very equivocal.

FIGURE VIII.
A large apical cavity (possibly a suppurating hydatid cyst), subsequently evacuated by air compression.

## ILLUSTRATIONS TO THE ARTICLE BY DR. J. F. MACKEDDIE.

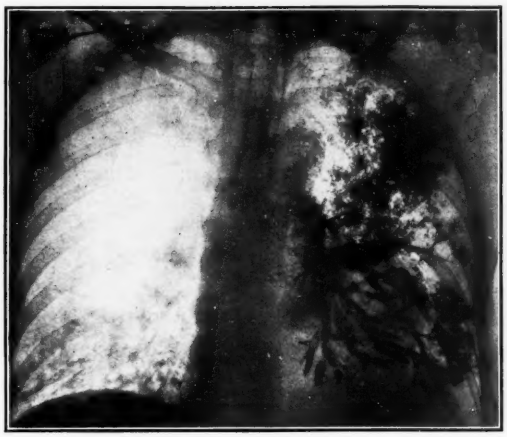

FIGURE IX.
An interesting type of general bronchial dilatations rather than terminal expansions.

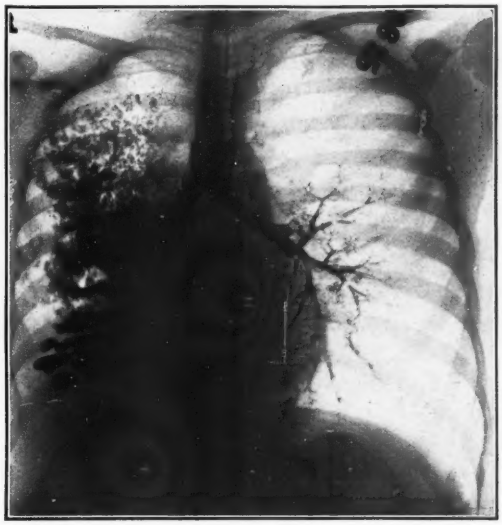

FIGURE X.
A fairly advanced cylindrical bronchiectasis.

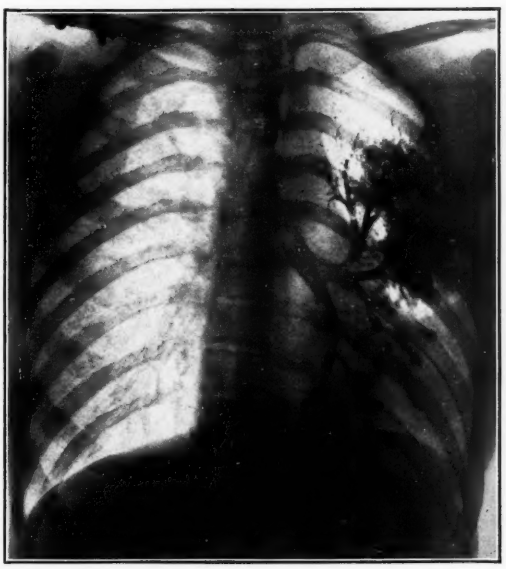

FIGURE XI.
Another type of discrete saccular bronchiectasis.

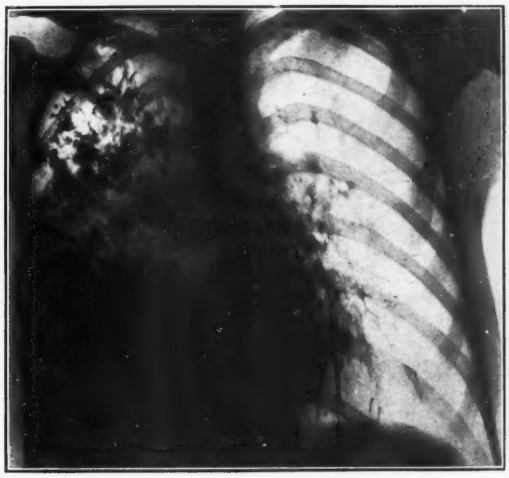

FIGURE XII.
A badly damaged lung with three distinct abscess cavities (after teeth extraction); successfully compressed.

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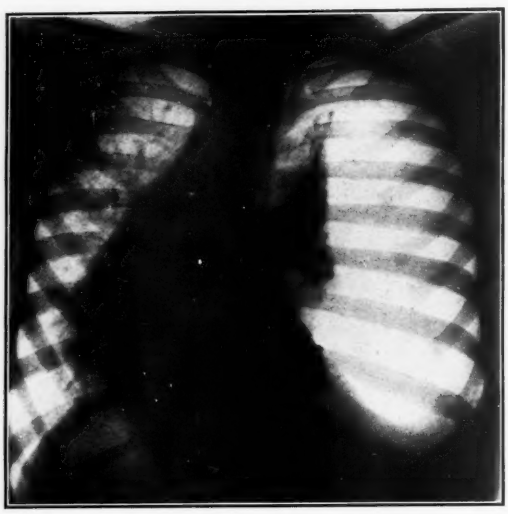


FIGURE XIII.
Basal cavitation (bronchiectatic) well compressed by air.

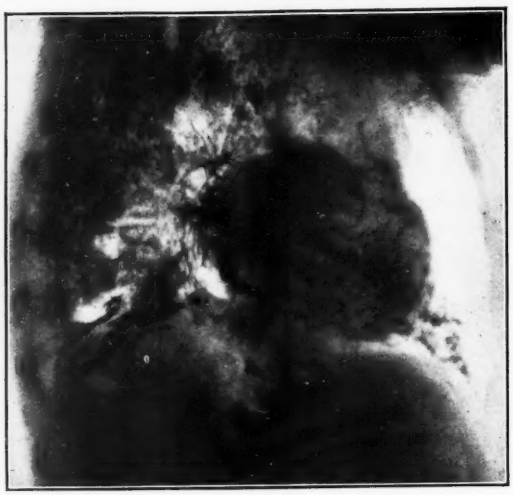


FIGURE XIV.
A rather uncommon picture of an enlarged heart due to chronic pericarditis.

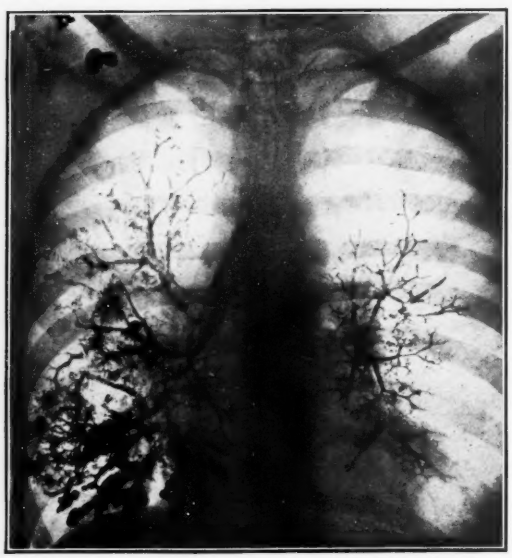


FIGURE XV.
Typical "clubbing" type of bronchiectasis.

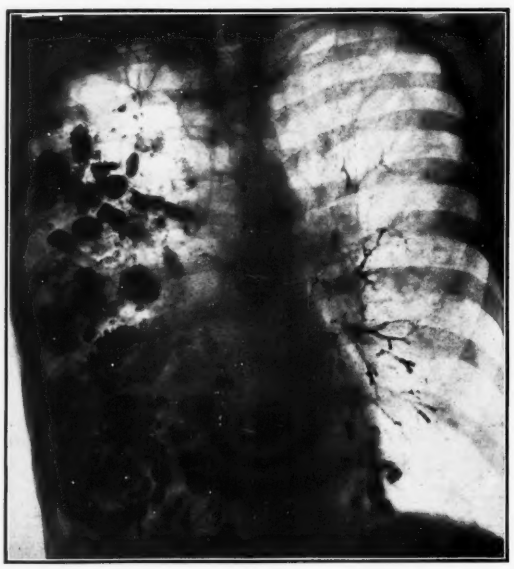


FIGURE XVI.
Probably an example of "Grawitz" type of general unilateral bronchiectasis. Compression by air was found to be impossible.

## ILLUSTRATIONS TO THE ARTICLE BY DR. J. F. MACKEDDIE.

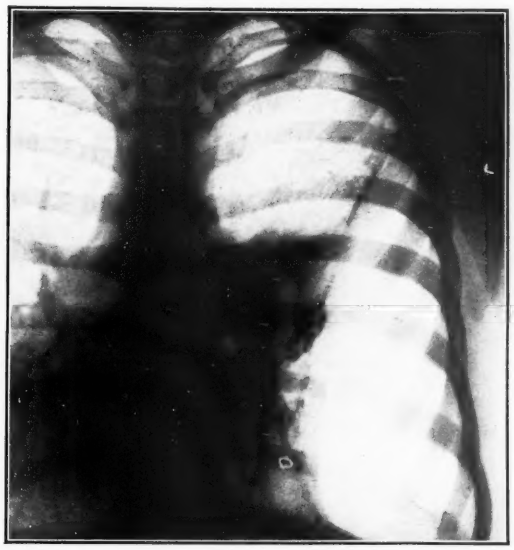


FIGURE XVII.
An abscess cavity partially collapsed by air, with the remains of lipiodol still showing.

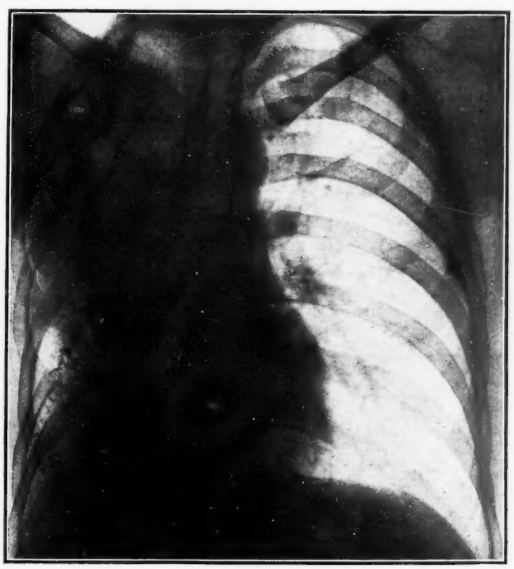


FIGURE XVIII.
A right thoracoplasty where lipiodol shows the reason of only qualified success. Quite a considerable amount of lung is uncompressed, with cavitation (from Dr. Downie's clinic).

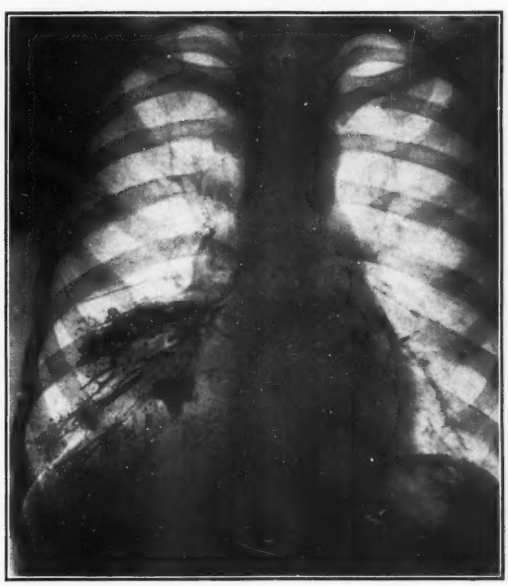


FIGURE XIX.
Small abscess whose position lent itself to easy filling by the supraglottal method of Singer.

## ILLUSTRATION TO THE ARTICLE BY DR. GARNET HALLORAN.

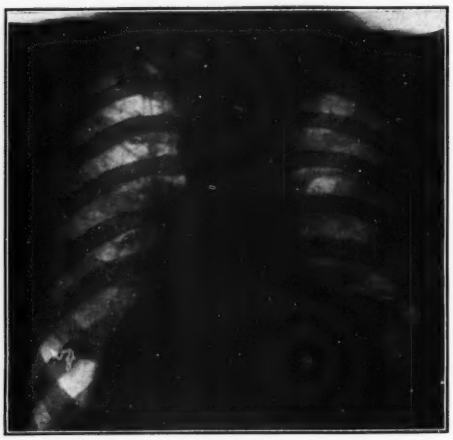
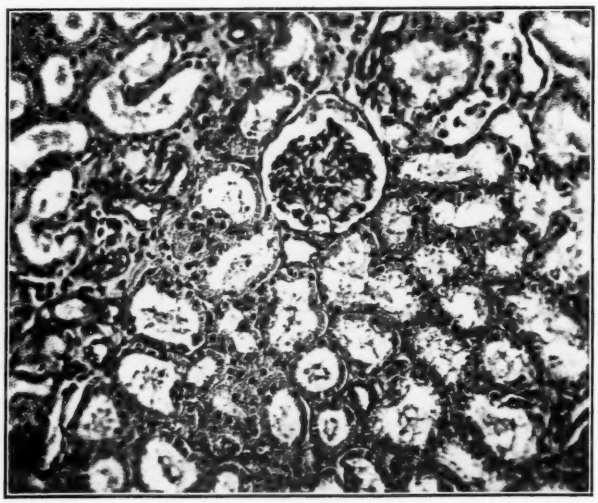


FIGURE II.

Skiagram of patient's chest. Note marked dulness in the right cardio-phrenic angle, suggesting bronchiectasis, and slight elevation of the right diaphragm with relative under-aeration of the right lung.

## ILLUSTRATION TO ARTICLE BY DR. S. F. McDONALD PUBLISHED IN THE ISSUE OF OCTOBER 10, 1931.



Photomicrograph showing damage done to the tubules while the glomeruli remain to a great extent unaffected. (Through an unfortunate oversight this illustration was omitted from the issue of October 10, 1931.)

## ILLUSTRATIONS TO THE ARTICLE BY DR. L. J. CLENDINNEN.

FIGURE I.

FIGURE II.

FIGURE III.

FIGURE IV.

## ILLUSTRATIONS TO THE ARTICLE BY DR. L. J. CLENDINNEN.

FIGURE V.

FIGURE VI.

FIGURE VII.

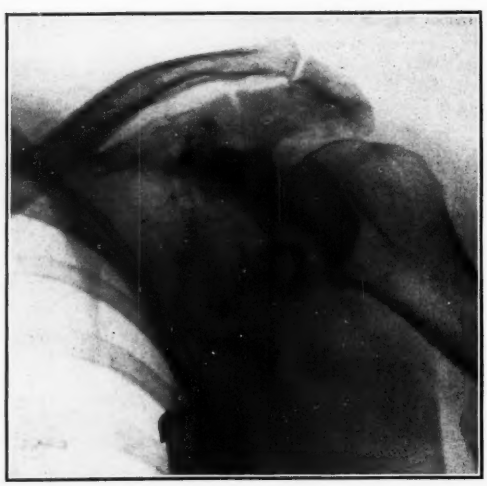


FIGURE VIII.

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## TRAUMA AND THE HEART.[1]

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*Honorary Physician, Royal Prince Alfred Hospital, Sydney.*

A DISCUSSION of trauma in relation to visceral organic disease was suggested in the first place by the many problems which arise, especially in the courts of law, in relation to the effects of the accidents and stresses of industrial life in causing or aggravating organic disease. We have discussed on many occasions, indeed *ad nauseam*, the effects of injury on functional disturbance of the nervous system, but little attention hitherto has been given to the question, more difficult in some respects, of how far organic disease is affected by trauma. In this study one turns to the text book in vain, except in the more obvious conditions of direct wounds of viscera. It is not with these that I intend to deal in opening this discussion, but with the more obscure questions of the effect of trauma on organs which are not exposed to external violence. There are many whose main interest is in diagnosis and treatment rather than in ætiology; but the more we know of cause, the more competent and intelligent we are likely to be in treatment. This discussion has therefore a wider clinical interest than the merely medico-legal one.

I propose here, for the purpose of the discussion, to expand the meaning of "trauma" beyond that of a morbid state produced by wound or external violence and to make it embrace the effects of physical work, prolonged or severe, following in this regard the practice of the courts.

I have heard the most diverse views expressed on the effect of work and "strain" in the causation of heart failure, in precipitating the rupture of blood vessels like the aorta and the cerebral arteries, in producing intestinal obstruction and rupture of bowel ulcers. The most far-reaching results have been assigned to actual injury as the cause of diabetes or of so reducing the resistance of the injured person that he is a ready prey to such organisms as the pneumococci and the streptococci.

I have heard it suggested, apparently seriously, that the mere act of going to bed for a few days was a contributory cause of sudden death from heart failure.

One suspects that often the view expressed is merely a "popular notion" accepted without thought and on no firm scientific or clinical basis. In seeking for an answer to many of these problems, it is necessary to refer them back to first principles and draw on the store of one's own experience.

From the very wide range of problems which suggest themselves, let us consider the effect of physical work, moderate or severe, in causing or precipitating failure of the heart.

There are some who believe that the healthy heart can be caused to fail by excessive exertion, a condition of "heart strain." The clinical evidence of this is often said to be an acute dilatation of the heart arising solely from overwork. The belief is widely held that in the diseased heart failure can be brought on by extreme exertion causing an acute dilatation. Some colleagues, for whose opinion I have the utmost respect, are convinced that in the healthy heart of an untrained individual, excessive exertion will cause overstretching of fibres of the heart muscle, which will thenceforth be permanently damaged. Indeed, acute dilatation by overstretching of the muscle fibres, healthy or diseased, is held by many to be a not unusual event.

Consider first the response of the healthy heart to exercise. Exercise may be, as Hill has pointed out: (i) Severe effort of brief duration, such as is involved in lifting a heavy weight; (ii) slight effort continued over long periods, as, for example, in industrial occupations; (iii) moderate or severe effort continued over fairly long periods, such as in a race from one to three miles or more. In the second type the fatigue is predominatingly of psychological origin, but in the first and third the physico-chemical changes in operation are to some extent similar, and involve not merely the heart, the voluntary muscles in use and the central nervous system, but all systems in greater or smaller degree. In lifting a heavy weight, the glottis is usually closed, the chest is fixed in inspiration and the heart distends till it fills the pericardium. "It is an experiment which may be performed time and again with impunity by healthy men."(1) Why can it be performed with impunity? Because the safeguards which Nature has provided for the heart are all in operation. These will be discussed later, but the fibrous pericardium calls for special mention at this point.

Many experiments by physiologists have abundantly demonstrated this fact, that sudden overdistension of the healthy heart from venous overfilling is prevented by the pericardium. Experiments with pericardial strips show that it is a relatively indistensible structure and even in the laboratory animal the work done may be suddenly increased to a degree equal to that which occurs

[1] Read at a meeting of the New South Wales Branch of the British Medical Association on July 30, 1931.

in the most extreme exertion and the heart is able to deal with it, if the pericardium is intact. The size of the heart may be suddenly increased experimentally by subjecting an animal to severe anoxæmia. It has been shown in monkeys and other animals with the pericardium removed, that during anoxæmia there is a much greater dilatation of the heart than in the intact animal. Indeed, if the anoxæmia is too rapidly produced, the animals whose pericardium has been removed, suffer an acute cardiac dilatation from which they do not recover, a phenomenon which does not occur with the pericardium intact. Also, if the vagus is stimulated until standstill of the heart occurs, the dilatation is considerably greater when the pericardium is removed than when it is intact.[2] Dilatation experimentally produced with a divided or removed pericardium may be of such a grade that rupture of the ventricular fibres may occur. In these experiments the degree of anoxæmia and of vagal stimulation is vastly in excess of that which is ever encountered clinically in the human being. The great weight of evidence, then, from the physiological laboratory shows that in the healthy heart, in times of stress, the pericardium alone is a safeguard against a harmful dilatation. And there is no clinical evidence that the healthy human heart can be damaged by sudden, severe, excessive work, such as occurs in lifting heavy weights, even to the utmost limits of the individual's powers.

The history of P.A., a miner, aged twenty-eight years, revealed no disease before his accident. While lifting a heavy beam overhead with another's assistance, his mate slipped and he had to support suddenly the weight of the beam with his arms above his head. He immediately felt a sudden pain in the left side of the chest near the position of the heart apex. This was accompanied by dizziness. He dropped the beam and sat down. In a few minutes he tried to resume, but was unable to do so. He thought he had "strained his heart" and went home and sent for his doctor. The latter, encouraged by the fact that some dyspnœa was present, confirmed the diagnosis, saying that there was acute dilatation of the heart. The patient was kept in bed for three weeks. He was seen six months afterwards, complaining of pain over the region of the apex beat, attacks of dizziness, palpitation and a capricious dyspnœa. On examination the pulse rate was 90 per minute, but there were no signs of organic disease in the heart or arteries. The electrocardiogram was normal, the Wassermann test on the blood gave a negative result. He was informed that his heart was normal, the court ordered the cessation of compensation payments and after a preliminary three weeks of light work, he was able to do the ordinary work of a miner without any discomfort or disability. When seen eighteen months later, he said that he had been quite free of symptoms and a careful examination of his vascular system showed no abnormality.

In this patient the original lesion probably was an injury to the left pectoral muscle and a cardioneurosis supervened, due to the fears of the patient and the incorrect diagnosis of his doctor. On questioning the latter, he confessed that his diagnosis was made because a very widely diffused cardiac impulse was present. He admitted that no overdistension of the veins of the neck was present, nor was the liver enlarged or the colour cyanotic. The clinical evidence was quite insufficient for a diagnosis of dilatation of the heart.

What are the clinical signs of pure dilatation, that is, dilatation unaccompanied by hypertrophy? It is clear that if a heart which was previously not enlarged, suddenly or quickly dilates, there is insufficient time for hypertrophy to develop. Obviously a "diffuse impulse" is quite insufficient for a diagnosis of dilatation. Although this physical sign does occur in dilated hearts, it is, by itself, no sign of abnormality. In the majority of patients showing this sign, dilatation is not present.[3] It is frequently found in healthy hearts beating at an enhanced rate and in normal young individuals.

There are many who consider it a simple matter to recognize the presence of dilatation by the position of the apex beat. If this is abnormally displaced, it is evidence (provided the heart is not displaced) of the size of the heart as a whole, and no decision is possible as to the relative amount of dilatation and of hypertrophy which is present. To assess the comparative degrees of hypertrophy and dilatation is rarely, if ever, possible. It is probable that in almost all instances where the heart is permanently enlarged, hypertrophy and dilatation are coexistent; to estimate the relative degrees of muscular growth and stretching in the living man is not possible; it is, moreover, unnecessary.[4]

Dilatation may be diagnosed as the predominant change only when the position of the apex beat has been observed on repeated examinations to move in the course of a few hours or days.

Percussion of the heart is of much less value than palpation. In some instances an approximate knowledge of the position of the left border of the heart can be obtained. When this is situated abnormally to the left, it is an indication of the presence of ventricular enlargement without reference to which ventricle is involved. It is not necessarily of itself indicative of dilatation or of hypertrophy of the left ventricle.

Percussion of the right border of the heart is of value only when the organ is displaced. It is difficult or impossible of accomplishment with any degree of accuracy and has little or no clinical value except in detecting or aiding to detect displacement of the heart. When the heart is not displaced and a right margin is recognized by percussion, it has relation, not to the ventricle, but to the right auricle, and we possess in the over-distension of the veins of the neck a much more reliable indication of enlargement of the right auricle.

A most valuable sign is the condition of the veins of the neck, since, when the right auricle is engorged, the veins of the neck become overdistended to a degree which is easily and definitely recognizable. This sign is of far greater consequence than any actual or supposed displacement of the right margin of cardiac dulness. "It is an unmistakable phenomenon, one of those emphatic and legible signs of which we possess no surplus."

I can recall no instance where the erroneous diagnosis of acute dilatation has been made in a previously healthy heart after sudden violent effort,

which was not an example of a cardio-neurosis usually supervening on a minor superficial injury.

Consider now the question of the effect of more prolonged and extreme exertion on the healthy heart. The trained athlete will run his race with increasing signs of cardio-respiratory distress and may run himself to exhaustion, ultimately even to unconsciousness. The untrained man will arrive at the exhaustion point earlier. Are these two in different case and does damage occur to the heart of one or other in such violent physical tasks?

The physiology of exercise is an exceedingly complex subject, but it may be of benefit to recall some of the known facts relevant to our study. During severe exertion the heart must increase its output from a rest rate of from three to five litres per minute to twenty litres per minute or more. Moderate exercise demands from eight to nine litres a minute. This increase in output is necessary to supply sufficient oxygen to the working muscles.

The healthy heart increases the volume per minute either by increasing the output per beat or by increasing the rate. Although increase in rate produces a smaller systolic discharge per beat, the total minute volume is increased by the rise in heart rate which occurs during exercise. If the heart is unable to discharge enough blood to produce a sufficient "head" of blood for the needs of the muscles, circulatory failure of cardiac origin is present. When the muscles are active, their vessels dilate. They need and can accommodate an enormously increased volume of blood. The return of blood to the heart is greatly increased by exercise, and the flow is facilitated by the pump action of the working muscles and the organs of respiration. The pressure in the great veins rises from these causes and also by direct action on the veins by the increase of carbon dioxide; the amount of blood pouring into the auricles is largely increased and the contraction of the heart muscle during systole becomes more effective. The force of contraction, therefore, is regulated by the volume of blood received from the veins during diastole. Up to a certain point, the greater the volume of blood in the heart, the more effective is the cardiac contraction. According to Starling's law of the heart, the force of contraction is determined by the amount of stretch of the muscle fibres at the beginning of systole. The optimum point of stretch is reached when a certain degree of dilatation of the heart occurs. This is physiological and is not recognizable by ordinary clinical methods. "As with every other muscle, if this point of optimum stretch is exceeded, the contractions begin to weaken and the systolic discharge will lessen. There is thus a physiological dilatation in which the volume of blood in the heart is increased until the fibres are stretched to their optimum length and a pathological dilatation in which this point is passed and in which the chambers may become pathologically dilated."

It is now accepted by physiologists that the point of optimum stretch of the muscle fibres is never exceeded in the healthy heart because Nature has provided adequate safeguards. The greatest clinicians are of the same opinion. Lewis has summed it up as follows:

> The circulatory organs are built to stand the strains which they themselves create; their reserve is often underestimated; it is the full supply of blood to the muscles which will fail, and not the heart, when the call is excessive. How comes it that in the horse, the most heavily strained beast of burden, chronic heart affections are almost unknown? *A priori*, the hypothesis of heart strain is not reasonable . . . If strain of the healthy heart exists, it is no more than a curiosity.

Still there are many who refuse to accept this conclusion. It is permissible, therefore, to review the foundation of this faith.

Attention has already been drawn to the protective effect of the pericardium. Although it appears that this is of great importance in sudden heart stress, there are clinical reasons for regarding other safeguards as more important in the protection of the heart muscle against damage from more prolonged severe work. Some instances have been recorded of partial or total absence of the pericardium. These conditions did not appear to produce any clinical symptoms and the response to effort was not limited, nor did the cause of death appear to be related to these congenital defects. Also, in persistent tachycardia occurring in a patient with apparently healthy pericardium, the heart may dilate pathologically if the tachycardia persists sufficiently long. Such an instance is referred to later.

But there are surer safeguards against pathological dilatation than the pericardium.

The physiologist has shown by experiment that the healthy heart is capable of dealing with the greatest increase of venous return and the utmost degree of diastolic filling which occurs during the most intense and prolonged effort.

Further, it has been demonstrated that before the heart is embarrassed or unable to cope with the venous return, there occurs such intense subjective distress that voluntary effort must cease. This is due to a rise in the hydrogen ion concentration of the blood and tissues due (*a*) to accumulation of lactic acid as the mechanism for oxygen intake fails, and (*b*) to the accumulation of carbonic acid as the mechanism for the elimination of carbon dioxide fails.[5]

There is in addition much clinical evidence to show that voluntary muscular work becomes impossible before any pathological dilatation of the heart occurs.

Many observers have investigated the size of the heart immediately after extreme exertion. Teleröntgenographic and orthodiagraphic means have been used extensively on healthy men, trained athletes, marathon runners, athletes out of training and men of no athletic prowess, and the overwhelming evidence is that the heart is smaller immediately after exercise, even though in some cases the individual was unconscious when the X ray picture was taken.

Everyone who has watched the finish of a boat race and has compared the state of the defeated eight with that of the victorious crew, will realize that there is a nervous factor at work in collapse after extreme exertion.

It is claimed by some that the heart muscle must be as susceptible to fatigue as any other muscle in the body. But the conditions under which the heart muscle acts are not precisely similar to those obtaining for skeletal muscle.

Starling[6] has pointed out that:

> The physiological condition of the heart is measured by the maximum pressure which it is able to produce in its cavities when it contracts, starting from a certain initial size and length of fibres. As the heart becomes fatigued, this pressure falls, so that the heart must dilate in order that each contraction shall produce the same maximum pressure as before. Fatigue of the heart is shown, therefore, not by failure to do its work, but by the fact that it can do its work only when it is undergoing considerable dilatation. Dilatation is therefore a measure of fatigue. What is often spoken of as the "tone of the heart" is really synonymous with physiological condition. A heart in good condition has a "high tone." It empties itself almost completely at each beat, even when receiving a considerable quantity of blood during diastole. A heart with a "low tone" is in the condition of a fatigued heart. It is dilated and, when it has finished contracting, still contains a large amount of residual blood.

The distress which terminates excessive muscular work is therefore not due to fatigue of healthy heart muscle, since such a grade of dilatation does not occur. The safeguards have intervened before heart muscle fatigue occurs. There are other considerations bearing on the question of exhaustion of heart muscle. Active tissues require more oxygen than inactive ones. Bainbridge has investigated the requirements of various types of tissue at rest and while active. This is shown in the accompanying table:

| Type of Muscle. | Oxygen used in cubic centimetre per minute per gramme of tissue. | |
|---|---|---|
| | While Resting. | Active. |
| Skeletal muscle .. .. | 0·003 | 0·08 |
| Unstriped muscle .. | 0·004 | 0·007 |
| Cardiac muscle .. .. | 0·007 | 0·08 |

From this it will be observed that skeletal muscle requires nearly twenty-seven times more oxygen during activity and the cardiac muscle nearly twelve times more oxygen. The requirements of the heart are such that the demands made upon the oxygen supply during activity are proportionately less than half of those made by the muscles.

The coronary circulation which is responsible for this is increased by the rise in pressure of the aorta which occurs during exertion; and dilatation of these vessels (and increased flow therefrom) is produced by two of the metabolites, namely, carbonic acid and lactic acid, which are known to be produced from the contraction of cardiac muscle. In addition, in conditions of stress, the presence of adrenalin in the blood produces dilatation of the coronary vessels.

Therefore, there is ample provision to postpone the exhaustion of cardiac muscle resulting from exertion.

Are there any conditions known clinically in which exhaustion of apparently sound cardiac muscle occurs?

A man, aged forty-two years, was attacked with paroxysmal tachycardia of classical type. He had suffered from two short attacks previously, but at all other times no symptoms or signs of heart disease were present. A fixed heart rate of 200 a minute, suddenly initiated, persisted for eleven days, when the heart showed signs of dilatation. The apex beat moved outwards to the anterior axillary line in a period of thirty-six hours, marked overdistension of the veins of the neck appeared. The patient became cyanotic, the liver enlarged and œdema of the feet developed. There were undoubted signs of dilatation occurring in a heart previously of normal size. After a few hours the paroxysm suddenly ended and in a few hours the heart returned to normal size and the venous congestion disappeared. The patient was able quickly to resume his normal habits of life and has remained well for several years since. No symptoms or signs of cardiac disease are now present. His heart muscle is apparently undamaged. In this patient the increase in heart rate was not due to the demands of increased work, the voluntary muscles were relatively inactive, there were presumably none of those conditions present or none of those safeguards operating which we have seen to obtain in severe muscular exertion.

It seems that this was an instance where a heart with apparently healthy muscle was caused to overact grossly and, as a result, ultimately became exhausted. If this interpretation be correct, it sheds light on the amount of overaction necessary to produce such a degree of exhaustion as to produce effects clinically recognizable. It also shows that the healthy pericardium will not prevent dilatation of the heart if the pressure is applied gradually.

The physiological and clinical evidence is overwhelming that the healthy heart cannot be injured by exertion, however severe.

In studying the effect of physical work on the healthy heart, one can draw on the results of much experimental work. But it is not so when the question of the diseased heart is considered. The great difficulty is that the condition of the muscle in the diseased heart is not a constant. The response of the diseased heart to exercise depends on the factors already considered in the healthy heart, but altered by the diminished power of contraction of the diseased heart muscle. The latter factor is variable and difficult of calculation.

Heart failure may occur gradually or suddenly. By sudden heart failure is meant the appearance suddenly or within the course of a few hours of evidence of failure in a heart which was previously competent, or of the striking increase of the symptoms of failure in a heart which had already shown a lessened reserve. Sometimes this occurs after the performance of some physical effort of a type which previously had been performed without distress or after an effort greatly in excess of the usual. If some individual act, such as lifting a heavy weight, should cause the onset of heart failure, the symptoms of failure occur at once. It is pertinent, therefore, to consider the conditions under which

sudden failure occurs. In this it is useful to discriminate between failure with abnormal and with normal rhythm.

### ARRHYTHMIA.

Arrhythmia is undoubtedly the commonest cause of sudden change from competence to failure and, in the great majority of cases, auricular fibrillation is the cause.

In 1922 Mackenzie pointed out that 60% or 70% of all cases of serious heart failure owe the failure directly or indirectly to auricular fibrillation or have the failure aggravated by its presence, and it is well recognized that effort may provoke the onset of this rhythm.

Reid says that:

> Although the onset may occur spontaneously or follow psychic states, such as fright or anger, he has observed patients in whom the attack commenced during an unusual exertion, such as pumping up a tyre or during retching. It is not unusual to have this arrhythmia develop in patients who may have had organic heart disease for years, during a fit of coughing in an acute respiratory affection, such as bronchitis. It is conceivable that in some of these instances it may be the accelerated heart rate which supplies the rapidly repeated stimuli to the auricular muscles, and that some one of these stimuli may happen to follow its predecessor at just the interval that is necessary to initiate a circus movement.[7]

Mackenzie states:

> The patient's description of his limitation is helpful, especially when he describes the distress which limits his power to respond to effort. This is usually distress in breathing. The onset of the distress is often so accurately described and so characteristic that while making an effort such as going rapidly up a hill or running, or lifting a weight, the patient became conscious of his heart running away, or he suddenly became breathless. Every case of "heart failure" with respiratory distress from what is called "heart strain" which followed some extra effort that I have seen, could be attributed to the onset of auricular fibrillation or its allied condition, flutter.[8]

Auricular fibrillation is prone to occur when the heart has been the subject of chronic inflammation (as in chronic rheumatic myocarditis) or of the effect of a specific toxæmia (as in exophthalmic goître) and in certain conditions in which the blood supply of the heart is impaired. There is evidence to show that it occurs particularly where arteriosclerosis is diffuse and has been gradual in its development, so that the smaller branches of the coronary arteries to the auricle are affected.[9] There are some who maintain that heart failure from auricular fibrillation may occur in the healthy heart after trauma, such as severe exertion or electric shock,[10] but, on the evidence, it is difficult to accept this view. It appears certain, however, that occasionally a heart which has previously given rise to no symptoms of disease, may develop auricular fibrillation immediately after exertion.

Once auricular fibrillation has been initiated in a heart which is so diseased as to be ready for it, the occurrence of dilatation and congestive heart failure is easy to understand. Occasionally other forms of arrhythmia determine the sudden occurrence of failure. Among such, auricular and ventricular tachycardias are to be mentioned, but these do not occur under circumstances which suggest that exertion plays any part.

It is important to recognize that the rapid dilatation of the heart which may occur under these circumstances is secondary to the arrhythmia. The literature of heart disease today and my own experience convince me that many instances showing the physical signs of sudden "acute dilatation of the heart" occurring when that organ is organically diseased are due to an arrhythmia, the change in the heart mechanism sometimes having passed unrecognized. It is an old notion that dilatation of the heart causes an increase in its rate. The facts are rather the reverse, namely, that an accelerated rate sometimes induces dilatation in an unhealthy muscle.

It is clear, therefore, that heart failure frequently occurs through the initiation of an abnormal rhythm. Statistically this type of heart failure is the most common. It occurs sometimes after exertion. Hearts whose muscle is so diseased that they are ready to fibrillate and which will inevitably do so anyhow, may need but little stimulus to provoke this change. The stimulus may be provided by a host of conditions, one of which is exertion. There is reason to believe that, with few exceptions, all instances of sudden heart failure following some severe individual act of exertion are instances where the initiation of a new rhythm has determined the failure. The exceptions are those instances where effort induces failure of a heart muscle suffering from active infection. Here failure may occur with normal rhythm.

### FAILURE WITH NORMAL RHYTHM.

By normal rhythm is meant that the auricles are responding to the impulses arising naturally in the sino-auricular node. This definition includes auriculo-ventricular block and sinus rhythm with occasional extrasystoles, but excludes auricular fibrillation, auricular flutter and paroxysmal tachycardia.

Heart failure may arise or suddenly increase without disturbance of the normal sinus rhythm, and it is important to examine the part played by effort in such instances.

Sudden heart failure with normal rhythm occurs in two conditions, namely, coronary occlusion and during acute infections.

#### Coronary Occlusion.

The recently acquired knowledge of coronary disease has shown that the occurrence of occlusion to a branch of the cardiac artery produces sudden or immediate death in but 50% (roughly) of patients. The 50% who survive, who may or may not have had symptoms arising from their cardiac disease before the occlusion, suffer thereafter from a marked diminution of the reserve force of their hearts. In most instances the disability is permanent. I am convinced that occlusions of small branches of the artery occur and mark the commencement of a period of increasing heart failure,

although the symptoms at onset may not be of the degree of severity which usually marks the occurrence of infarction. My experience of such phenomena, verified by clinical and electrocardiographic evidence, suggests that this occurs particularly in elderly people in whom the anastomosis of the coronary vessels is at its greatest development. The following cases may be quoted as examples:

CASE I: R.J., aged sixty-eight years, a merchant, while seated at breakfast, felt severe pain in the upper part of the abdomen and beneath the sternum. He was slightly breathless and shortly afterwards fainted. His wife noted definite pallor and that his pulse was "fluttery" and weak. The pain and vomiting continued for thirty-six hours. At this time clinical examination of the heart revealed no definite sign of abnormality. The pulse was regular and of low tension. An electrocardiogram taken forty-eight hours after onset of symptoms revealed the characteristic changes of coronary occlusion. After two weeks in bed without symptoms he insisted, against advice, on resuming a life of some activity. Some extrasystolic arrhythmia frightened him back to bed for a few days, but he has since (for eight months) led a life of very slight activity, his response to effort being very appreciably less than it was before the onset. Now he is unable to walk at a moderate pace on the level.

In this patient a definitely recognized coronary occlusion produced a sudden change from competence of the heart to failure, although it did not cause the usual grave syndrome.

CASE II: A.K., aged seventy years, a quarryman, was in good health until one morning, while doing his usual work, breaking stone, drilling for blasting and barring down, he had a "heart attack." He had turned over a large stone with the help of another man without discomfort. He had bored two holes with a pick, but on putting in the wedges his head suddenly went round and he felt that he was going to faint. He did faint, and on coming to, vomited. He was in hospital for three weeks. Since leaving hospital he has been weak and very short of wind on walking any distance. Substernal pain is present on the slightest exertion.

On examination six weeks after onset there was found to be marked enlargement of the heart, and at the aortic and mitral areas a systolic murmur was present. The pulse was regular and the blood pressure reading was 175 millimetres of mercury systolic and 95 millimetres diastolic; the vessels were much thickened and calcified. The electrocardiogram revealed left ventricular preponderance only.

In this patient the cause of the sudden increase of cardiac disability is not proven beyond doubt. The physical examination does not explain it. One is forced to form a conclusion from the history of onset. I believe that here a coronary occlusion occurred and that its onset coincided with but was not due to exertion.

In patients whose history shows the sudden occurrence or a rapid increase of symptoms of cardiac disability caused by coronary occlusion, the pulse, in spite of temporary arrhythmias, is usually regular in rhythm.

In the published case records of patients with coronary occlusion, such as those of Levine and Parkinson, there occur several instances of similar type. It is probable that heart failure occurring or increasing suddenly without marked disturbance of rhythm is due much more often than is usually recognized, to coronary occlusion, sometimes not of major degree.

It is a sound generalization that in all instances in which the onset of heart failure is with substernal pain, fainting or collapse, followed by congestive failure with normal rhythm, the cause is occlusion of a main branch of the coronary artery.

I am of the opinion that exertion plays no part in causing or aggravating heart failure from this cause.

### Active Infections.

The effects of active infection on the heart muscle in such conditions as diphtheria and pneumonia, and in even less serious ones, are very familiar. Mackenzie was the first to lay stress on the fact that the explanation of the failure of the apparently healthy hearts of some patients after exertion lay in the presence of an actual infection.

It is also a common clinical experience that a diseased heart which has previously been competent, will fail after an illness of infective origin, such as severe influenza, bronchopneumonia *et cetera*. Failure in these circumstances may occur with normal rhythm. It is easy to understand the loss of contractile power that such muscle fibres show.

The literature of heart failure with normal rhythm is surprisingly sparse. Parkinson and Clark-Kennedy[11] investigated clinically the details of 100 patients with severe heart failure with normal rhythm. In 48 instances a necropsy was done. Patients below the age of twenty years and over the age of seventy were excluded, also patients suffering from acute infections, bacterial heart disease or rheumatic carditis. No other method of selection was used. As an indication of the relative infrequency of this combination, it is of interest to note that it was only with great difficulty that they were able to collect this series in various large London hospitals in a search extending over two years.

From their very careful study Parkinson and Kennedy concluded that hearts which failed with normal rhythm were those (i) in which a focal coronary lesion, such as occlusion of a main coronary branch, had occurred; (ii) in which there was an acute infection (acute rheumatism, bacterial endocarditis, diphtheria, pneumonia *et cetera*); and (iii) in which failure was largely determined by gradually developing mechanical factors, the auricular muscle not being primarily diseased; for example, uncomplicated high blood pressure, chronic pulmonary disease, congenital malformations and gross aortic incompetence (syphilis). In the latter type sudden heart failure does not occur.

Parkinson and Kennedy assert that the hearts which fail characteristically with normal rhythm (apart from the types already discussed in which coronary occlusion or active infection is present), are those in which the main pathological change is not generalized in the heart muscle, but in the aorta (as in syphilis), in the systemic blood vessels (as in hypertension), or in the lungs (chronic conditions such as emphysema and bronchiectasis). In their series of failure with normal rhythm was

included a group of "primary myocardial" types in which no clinical evidence of the cause of heart failure was present. In such of these as were examined *post mortem*, the cause was found to be either previous high blood pressure (systemic cardiovascular hypertrophy) or occlusion of a main coronary branch. In none was heart failure due to chronic myocardial inflammation or degeneration. No example in the whole series was seen of failure with normal rhythm from non-rheumatic primary myocardial disease or degeneration.

There is support to these views from other investigators.

#### Hypertension and Generalized Arterial Disease.

It has been stated that heart failure with normal rhythm is the rule in hypertensive heart disease. Janeway, Clifford Allbutt, Galavardin and Cabot all support this view. Allbutt was of opinion that when arrhythmia occurs, it is the heart muscle that is primarily at fault.

The pathological evidence is clear that when the main change is in the systemic vessels outside the heart, the myocardial changes are those of hypertrophy and not of degeneration. Cabot in reviewing the results of a series of over 1,000 necropsies on patients dying with enlarged hearts, a great number of which were associated with arterial disease and hypertension, states that most of these showed no myocarditis and that after correlating the pathological and clinical evidence, myocarditis played no demonstrable part in producing failure. Clawson[12] arrived at a similar conclusion, namely, that myocardial strain (hypertensive or non-hypertensive) is not a cause of myocardial fibrosis.

#### *Syphilis.*

Many authors have commented on the infrequency of arrhythmia in cardiovascular syphilis. Although the treponema is found in the heart muscle of the syphilitic, no extensive degeneration and certainly no marked functional damage is present. Indeed, the heart muscle function in syphilitic aortitis is usually good. Cardiac syphilis usually means syphilitic aortitis, and myocardial changes are secondary to this. Myocarditis is slight.

#### *Chronic Pulmonary Disease.*

Here also necropsies on patients who have died of heart failure reveal that the myocardium shows slight, if any, histological degenerative changes.

#### *The Myocardium.*

The common factor in these types is the presence of a myocardium, certainly hypertrophied but not markedly degenerate from old inflammation or nutritive changes. Where hypertrophy is present there has been necessarily a good coronary blood supply. It is suggested, therefore, that if heart failure occurs with normal rhythm, the primary cause is not in degenerative changes in the myocardium. Yet hearts of this type do fail ultimately. Clifford Allbutt, in discussing the cause of heart failure in uncomplicated hypertensive heart disease, was of the opinion that when increasing resistance imposes an insuperable load on the heart, failure supervenes as a confession of "cardiac defeat." Since there are not considerable degenerative changes in the myocardium, the ultimate cause of failure must be an insufficient blood supply, a failure of the coronary circulation to satisfy the demands of a hypertrophied musculature. Is the heart failure which occurs in such conditions always a gradual affair or does it sometimes occur suddenly without fibrillation or occlusion? And if it does occur suddenly, does this happen after severe exertion? I can supply no positive answer to these questions, but I believe that failure in such conditions is always gradual and unrelated to effort. The published case records are not detailed enough to enable an opinion to be formed, but they indicate that failure is gradual. I have referred to my own records of patients in whom the evidence is complete, but these are insufficient in number. I suggest that this is a point on which more extended clinical observations are necessary. Indicating the clinical caution necessary, I give an instance:

C.H., aged sixty-three years, a general labourer, when lifting a log noticed a pain across the heart. He continued work that day, using a crosscut saw, but was unable to do any lifting. Two days afterwards he discontinued work because of pain and dyspnœa. He was kept in bed for two weeks, and after another week resumed work, but was unable to continue.

On examination twenty-five days after onset of symptoms he was found to be dyspnœic after mild exercise, the heart was enlarged, the pulse was quite regular, the blood pressure 170 millimetres of mercury, systolic, and 110 millimetres of mercury, diastolic. There was slight œdema of the legs and congestion of the lungs. The electrocardiogram showed right branch bundle block and the *R-T* interval was of the type indicating coronary occlusion. The patient lived for twenty-two months with a gradually lessening response to exercise, and died of congestive heart failure.

This is the history of a patient who suffered from acute heart failure with normal rhythm, in whom the opinion might have been given excusably that overstrain of heart muscle fibres had occurred. The electrocardiogram, however, gave conclusive evidence that the cause of failure was coronary occlusion.

Should further clinical investigation prove that severe heart failure with normal rhythm is always gradual and does not occur or suddenly increase after exertion, it will indicate that exertion does not produce a sudden "strain" of hypertrophied heart muscle if its vascular supply is good. The effect of continued or repeated exertion which is excessive for the condition of the hypertrophied heart muscle, will be referred to later.

The changes found in the heart at necropsy, of course, are of great importance, but it is not always possible to deduce from them the disorder of function which has caused the death of the patient. In no condition is this so true as in the fibroid heart, so well recognized by the pathologist, but so incapable of recognition by the clinician.

Degeneration of the heart muscle fibres occurs after inflammation, as in rheumatism, and similar

fibrous changes arise from generalized arteriosclerosis (whatever further ætiological mysteries may be hidden behind this familiar term); and other causes, unknown for the most part, may give rise to fibrosis. The view is widely held that when exertion appears to damage permanently the efficiency of the degenerate heart muscle, an actual strain of the muscle fibres has occurred through stretching of the fibres beyond the limits of their elasticity. It is thought by some that this leads directly to a pathological dilatation of the heart, which is the primary cause of failure. It is probably true that the dilatation (when it occurs) is secondary to diminished contractile force, that is, to inability of the ventricular muscle to expel a sufficient quantity of blood during systole. Necessarily the heart dilates, and only by dilatation can its force be increased. Dilatation does not arise because the inflow of blood distends the passive heart beyond its normal limits. The normal heart is completely relaxed during diastole, the pericardium alone limiting its size. There is no reason to believe that it is different with the diseased heart. Any loss of elasticity on the part of its fibres would not influence its diastolic filling. But diminution in the contractile force brings it about that the ventricle is not sufficiently emptied and gradually more and more blood collects in the cavities of the heart and a gradual dilatation occurs. This is pathological dilatation and it is usually gradual and not rapid. If by rest and treatment a halt can be called, the heart muscle may to some extent recover, but if the process goes on, a point is reached where the contractile force of the fibres is permanently and progressively damaged. This change was considered by Clifford Allbutt to be of the nature of a permanent molecular alteration of the fibre. This explanation is a purely hypothetical one, just as the suggestion of Bainbridge that the change is a chemical one. These theories have no basis of proof at present, and are not needed to explain the failure produced by auricular fibrillation and coronary occlusion. In these conditions it is clearly diminished contractile force and not overstretching which is the cause of the congestive heart failure. The application of these theories to failure during active infection has much to commend it on clinical correlated to histological evidence. Is it satisfactory for the diseased heart in which fibroid changes are present? It is at first sight the most satisfactory explanation, but there are some considerations which make me hesitate before accepting it as the root of the matter.

It is a well known fact that at the *post mortem* examination of patients who have died of such diseases as cancer, there has been found a very extensive replacement of muscle tissue by fibrous tissue in the walls of the heart, despite the fact that there has been a relatively good cardiac function to the end of life. Cabot[13] has analysed the case histories and necropsies of a series of 91 patients who died with definite and extensive fibrous changes in the heart wall, and found that death occurred from heart failure with congestion in only three patients who did not show coronary disease or infarction.

Evidence of this kind suggests that the real cause of the inability of the heart to maintain the circulation was rooted in a deficient blood supply rather than in a permanent so-called metabolic or chemical change in the fibres, due to overstretching. Or that, if permanent overstretching of fibres has occurred, it was made possible by the nutritive changes dependent on an inefficient coronary system. In laboratory experiments Patterson and Starling have shown that interference with the nutrition of the healthy heart muscle immediately diminishes the minute volume of the heart.

Whatever the truth of this matter may be, I believe that the sudden occurrence of rapid increase of the signs and symptoms of congestive heart failure is caused in the great majority of instances (when no active infection is present) by a change in the heart rhythm (usually auricular fibrillation) or by the onset of coronary occlusion, and that the important change in the diseased heart muscle fibres is dependent on deficient blood supply causing lessened contractile force. Over-exertion may play a part in causing arrhythmia, but not in the vascular conditions, and Mackenzie's statement made seventeen years ago still holds good.

There still remain to be considered those instances in which sudden heart failure, not of congestive type but with angina, appears to date from or be greatly accentuated by unusual exertion. A discussion of this matter would involve the consideration of the whole question as to the cause of precordial pain. There is not time for this and the question has been thoroughly dealt with at a recent meeting of the Section of Medicine. I have already dealt with congestive failure appearing after substernal pain.

### Gradual Heart Failure.

Frequently heart failure is a slow process in which the capacity of the heart to maintain the circulation gradually diminishes, the reserve force of the heart gradually disappears and heart failure leads on gradually to death. The reserve force of the heart is measured by the capacity of the ventricles to maintain normal output in spite of a raised arterial pressure and to increase output when the venous inflow becomes larger. Although the individual may live a guarded life, avoiding such exertion as would materially raise the arterial pressure or increase the venous inflow, yet the heart will fail ultimately if the contractile power of the cardiac muscle is waning. The circulation gradually slows, œdema appears, followed by the other evidences of failure. Such a train of events occurs occasionally, being seen particularly in the aged. In such circumstances as these exertion plays no part in failure and therefore we are not concerned with it. If such a patient lives a more active life, the progression of heart failure to death, though gradual, occupies a shorter time, according to the

degree of effort which is exerted. As Levine[14] says:

> The question may be regarded in the light of a mechanical engine which cannot deteriorate through disuse (for the heart is always contracting anyway), but the life of which will be shorter the more it is used. This is not unlike the general conclusion Raymond Pearl came to in studying fruit flies when he stated that "in general, the duration of life varies inversely as the rate of energy expenditure during its continuance."

And the energy expenditure of the heart is dependent on its vascular supply.

Sometimes an individual in such circumstances first becomes aware of a definitely diminished cardiac capacity after some exertion. In patients in whom a gradual deterioration of function is occurring it is not surprising that the first distress should come at a moment of severe exertion; at such a moment the reserve power is called upon and found wanting. If no active infection is present, if no fibrillation of the auricles occurs at such a time, and if the coronary circulation is good, I believe that the exertion produces no permanent damage to the heart muscle.

### SUMMARY.

1. Severe exertion causes no damage to the healthy heart.

2. The diagnosis of "strain" of the healthy heart is frequently made in error, the condition being a neurosis.

3. When the apparently healthy heart does fail after exertion, failure is due to lack of contractile force of the heart muscle fibres injured by active infection.

4. Exertion may cause the diseased heart to fail if it precipitates fibrillation of the auricles.

5. Severe heart failure with normal rhythm occurs much less frequently than with arrhythmia.

6. Occlusion of a main coronary artery or branch may cause sudden heart failure with normal rhythm. Exertion plays no part in this.

7. A heart which is the subject of active infection may be caused to fail with normal rhythm by exertion.

8. The primary factor in failure of the diseased heart is lack of contractile force and not dilatation of the heart.

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## TRAUMA IN RELATION TO ORGANIC VISCERAL DISEASE.[1]

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"TRAUMA in Relation to Organic Visceral Disease" is a title admitting two lines of study, for we may consider the effects of trauma on the subjects of organic visceral disease, or we may consider trauma as a factor in the causation of visceral disease. In this paper we consider it sometimes from the one, sometimes from the other point of view. But although a rigidly logical method demands it, I am not going to spend time and trouble in finding a strict definition of the meaning of the term "trauma." We can do better without.

Apart from academic interest, it is eminently desirable that we should devote our best intelligence to the problems involved, for decisions have to be made by Workers' Compensation courts upon the issues of which depends the economic disposition of hundreds of thousands of pounds, and this inevitably reacts on the cost of living in the community. It is a short-sighted view which looks on it as a mere matter of profit or loss for the insurance companies. Everything is passed on.

The decisions of these courts often depend on the answers to medical questions. It is a great responsibility. The problems must be thought out honestly and carefully before we venture on an answer. Yet it is true that many medical men allow themselves to be deceived too easily by appearances. Courts are likewise liable (as experience shows) to be taken in by the seemingly obvious. And we are subject to an unfortunate weakness, common to humanity, a weakness that causes us to see or remember best what we want or what it is suggested to us to see or remember, and it makes us apt to be strangely blind to what does not favour our preconceived idea.

It is impossible to deal properly with the subject without reference to elementary physics, but nothing more than all of us have learned in the early part of the medical course, though it may not all be clearly remembered.

The material does not permit of a strict logical order in treatment. It will have to be taken more or less at random.

### Retention of Urine. Prostatic Hypertrophy.

Retention of urine is liable to occur in any man suddenly laid up with an accident, or even without an accident. I have heard this denied in evidence by medical witnesses. While retention from this cause is not confined to old men, it is particularly

[1] Read at a meeting of the New South Wales Branch of the British Medical Association on July 30, 1931.

liable to occur in men whose prostates are enlarged. Following the condition there may be serious effects, either from bladder distension or from the catheterism employed to relieve it.

#### Ruptured Bladder.

Rupture of a distended bladder involving the peritoneum may occur in an accident when a blow has been received on the lower part of the abdomen. It would be more liable to occur in a man with an enlarged prostate, for several reasons. First, because the bladder is more likely to be full, or nearly full; second, because when distended there is a larger target, and third, because, as all will probably agree, the bladder wall is more frangible.

There are two ways in which a force applied to the front of the lower part of the abdomen may burst a bladder. A hollow organ will hold the largest quantity when it is approximately spherical. If the bladder is squeezed back against the vertebral column, it will be distorted and become less spherical, and since liquids are practically incompressible, the hydrostatic pressure within is raised, and may pass beyond the accommodating or resisting power of the bladder wall, when it must rupture. This type would be seen in buffer injuries or when a wheel has passed over the abdomen.

But a violent blow may develop so much kinetic energy, when arrested more or less suddenly by the mass of the bladder, that it will rupture it, even when it is cushioned on intestine.

#### Train Accidents. Car Accidents. Falls from a Height.

A blow made by some moving object on the abdomen is equivalent to a blow by the abdomen on the object. We see this condition in train and car accidents when the vehicle collides with an obstacle and is suddenly stopped. The potential energy of the moving body, or moving mass of the bladder is translated into terms of work, and the work may be destructive. A fall from a height, if the height is sufficient to create enough kinetic energy, can produce a similar effect.

##### *Demonstrations by Experiment.*

By filling some such object as a toy balloon with water, we could exemplify all these ways of rupturing a bladder. For we could burst it: (i) by running a car wheel over it, (ii) by hitting it on one side with a golf stick, (iii) by throwing it against a wall, or (iv) by dropping it to the ground from a sufficient height.

Note as regards the second method that the more water it contains, the bigger is the target it presents, and note further that the more water it contains, that is, the greater its mass, the more readily will it rupture when thrown or dropped. When hit with a stick, the heavier the weapon, that is, the greater its mass, and the greater its velocity, the more destructive its effect. If it is thrown against a wall, the greater the velocity with which it hits the wall, the greater the effect. If it is dropped from a height, the further it falls, the greater its velocity when it strikes the earth, and the more destructive is the effect.

##### *Symbolic Expression of Physical Facts.*

This is greatly clarified by resorting to the mathematical expression of the physical facts. As stated in this way, the kinetic energy of a body of mass $m$ moving with a velocity $v$ is $\frac{1}{2}mv^2$. If this be brought to rest by an opposing force $F$ and the space traversed by the body while this force $F$ is acting is $s$, the work done against the force is $Fs$, and the kinetic energy equals $Fs$. But if the space traversed is small, which is approximately the case, or often so, in the organs of the body, then the kinetic energy does other work than altering the position of the organ. If the work done were to consist in changing the kinetic energy into molecular motion, it would become manifested as heat, but in the human body it would be manifested mainly in other ways, such as bruising, shattering, tearing and bursting of tissues and organs.

##### *The Kidney.*

Leaving now the bladder for the kidney, we can say with confidence that a hydronephrotic kidney would be more vulnerable to a blow or to a squeeze than would a normal kidney. By "squeeze" I mean an injury of the buffer type, not an increase of the hydrostatic pressure within the abdomen, which is no more damaging than the atmospheric pressure on our bodies.

When a kidney contains a small calculus, a blow may dislodge it into the ureter, causing colic. If the calculus is a large one, it will not be dislodged, but hæmorrhage may be produced. An X ray examination should reveal the calculus. If none appears, either it has been passed, or the condition was one of the passage of uric acid sand, not reasonably attributable to trauma.

#### Hydatid Cysts.

Being in Australia, we pass naturally from the bladder and hydronephrotic kidney to the consideration of hydatid cysts. In the lung these cysts have hardly any adventitious wall when they are small, and not much even when large. In the abdominal cavity they have a thin membranous adventitia, and in the liver the adventitia is much stronger.

No hydatid cyst can be ruptured by mere increase of the surrounding hydrostatic pressure. In a hydrostatic pressure, the force acts always perpendicular to the surface, and on each unit of surface it is everywhere the same in magnitude. There is, therefore, a perfectly even stress, and a liquid is practically incompressible. The contingencies and conditions formulated when considering ruptures of the bladder apply to hydatid cysts, wherever they are. What may burst the parasitic cyst may burst the adventitial covering. Following on ruptures of the cyst there may occur infection and death of the parasite. Or the contents of the parasite may escape and disseminate scolices and daughter cysts. As another result I may mention a case in which a blow ruptured a cyst into the liver

substance and caused biliary colic associated with hæmorrhage into the ducts, the blood filling also the gall bladder. There is what may seem at first an exception to the rule about an increase of surrounding hydrostatic pressure not causing a hydatid cyst to rupture. This occurs when a cyst of the liver has herniated through the diaphragm. In such a case, an increase in the intraabdominal hydrostatic pressure could, and does, cause rupture into a bronchus through adherent lung. It is not really an exception to the rule, because at the place where it bursts the pressure is less than the pressure behind it in the abdomen. If it were not less, it could not burst.

As to hydatid cysts of the lung, they are well protected against violence from without. The compression stress is a pure hydrostatic pressure, and therefore the cyst is evenly supported in that respect. A large proportion of hydatids of the lung rupture when small, which usually effects a spontaneous cure. The rupture must proceed from some change producing an uneven stress. I can imagine a local congestion producing either an effusion of serum or a small hæmorrhage into the delicate adventitious capsule, distending it or rupturing it, and pushing before it the parasitic cyst. In this way, the ectocyst of the parasite might be forced to rupture. But that is conjecture. However, if that, or something like it, is the mechanism, it is not produced by trauma, though I do not forget that I have given no definition of that term.

### Injuries of the Liver.

Let us turn now from cystic structures to the liver and spleen. These are solid organs, both of them rather brittle, and both of them may vary in size and in consistency under various conditions. The fatty liver is larger and softer and more vulnerable, not only on account of its extra size making it a larger target, but also because it ruptures more easily. In the domestic animals this tendency of an enlarged fatty liver to rupture has drawn special remark. The statements made as to the behaviour of a balloon filled with water apply just the same to a solid organ such as the liver or spleen, and the same mathematical statement applies. The only difference is in the varying resistance to structural damage. It may take more work in one case, or less in another, to do serious damage.

### Injuries of the Spleen.

The spleen, when of normal size, is rarely injured. But in countries where malaria is endemic, very large spleens may be found among the affected inhabitants, and in those countries ruptured spleen is relatively common. The large spleen offers an exposed target, and in falls its large mass greatly increases the kinetic energy, other things being equal, and so it is more liable to damage. And in a large spleen there is also a likelihood of compression when a fall occurs, because the mass of the body forces the organ against the ground in addition to its own intrinsic mass. Accidents of this kind come to the same thing as if the organ received simultaneously a blow from the front and a blow from behind.

Neither liver nor spleen can receive injury from heavy lifting or any other cause of increased intra-abdominal hydrostatic pressure. This cannot possibly cause anything else but a compression stress, and the organs are incompressible, being practically the same as water. Only a shearing stress can cause deformation in such organs, and such a stress cannot be exerted by a surrounding hydrostatic pressure.

### Injuries of Other Abdominal Organs.

We come next to injuries of the omentum, stomach, and intestines caused by violence. When the muscles of the abdominal wall are relaxed, or at any time if the kinetic energy of a blow is extremely great, then the contained viscera, whether hollow or solid, are deprived of the protection usually afforded by the tonic contraction and consequent rigidity of those muscles. With this protection gone, direct blows on organs are possible. But the most usual type of injury seems to be one caused by crushing, such as by buffer accidents, or by being run over, or the like. In these the injuries can usually be explained as due to crushing of the organ against the prominence of the vertebral column. But sometimes a portion of intestine, especially large intestine, may be held in what is at the moment practically an air lock, and a sudden compression could burst it. In old people, and in states of inanition, the large intestine is often as thin as tissue paper. It is reasonable to conclude that such people are particularly vulnerable to crushes involving the abdomen.

Most cases of rupture of the intestine are fatal, but omental injuries may be recovered from. It is quite possible that people who have recovered from such injuries may recover with adhesions that could at a later date be the cause of an intestinal obstruction.

#### *Perforation of Gastric and Duodenal Ulcers.*

From this we come to the consideration of perforations of gastric and duodenal ulcers. Some of us were surprised at the evidence, and greatly concerned at the decision in *Cleary v. Wilson Granite and Gravel Company.*[1] Only a complete cynic could remain undisturbed, for it showed that in a really scientific question like this, the court could not tell one kind of evidence from another. And yet it is the most competent and discriminating court in Sydney as far as medical questions are concerned.

Believing that something ought to be done about it, I had almost finished a paper on certain physical questions involved before I was asked to read the present one. The other paper has a slightly mathematical appearance, and is not very well adapted for reading to an audience, but perhaps it can be taken as a complement.

An ulcer is, more or less, a weak spot, less resistant to disruptive stresses than the normal tissue.

In forces tending to strain the wall of a viscus, the breaking strain is reached first at such a spot, and there it ruptures. We can conclude without hesitation that in cases where a direct and forcible blow is able to reach a dilated stomach, unprotected by the tonically contracted guarding muscle of the abdominal wall, then the force might cause perforation at an ulcer, its weakest point. Such a case is parallel to those I have mentioned as possible in connexion with a dilated large intestine. It is, however, only a theoretical possibility. I doubt if it has ever been recorded.

But, apart from the general intraabdominal pressure, intragastric pressure is controlled normally by muscular contraction of the stomach wall, and the same applies to the duodenum. The stomach contracts down on to its contents and forces them onwards as digestive processes may require. This is accomplished by the well known peristaltic movement of the stomach wall. One can add that the vertical depth of the gastric contents presents an addition to the hydrostatic pressure at the lower part of the organ. It has been abundantly proved that the ingestion of food or drink stimulates the gastric movements and increases intragastric pressure. It is this increased intragastric pressure that bursts through the weak spot and causes perforation. Eating and drinking is done during the waking hours, not during sleep. Digestion and gastric movement are believed to be at a minimum during sleep; therefore perforation during sleep is unusual. It is widely believed by physicians that strenuous exercise is not good soon after a meal, because it tends to inhibit digestion. If there is reasonable foundation for that belief, it would be rather paradoxical to expect an increase of peristaltic movements during active exercise, and due to the exercise.

In *Cleary v. Wilson Granite and Gravel Company* it was stated in medical evidence that muscular effort increases the peristaltic action of the stomach and intestines, and that this would increase intragastric pressure. The stomach was adherent to the liver by an ulcer, and it was stated that this increased intragastric pressure would tend to tear it away, and so cause rupture.

It would seem that others have discovered that muscular effort increases peristaltic action, for in *Ryman v. Municipal Council of Mosman* medical witnesses stated that muscular effort caused increased peristalsis of the intestine, thereby tending to produce intestinal obstruction in a person predisposed by the presence of an old intestinal adhesion.

Now, if it is true that muscular effort causes increased peristalsis, these gentlemen, all of them, have found something very far-reaching indeed. We all know that appendicitis can be brought on by an attack of diarrhœa, or by a mere purgative. Now we shall be able to explain appendicitis as due to the work a man was doing. What a vista that opens up!

We are finding that *Cleary v. Wilson Granite and Gravel Company* is a case that gives us a text for discussion at all points. In the case in question the site of the ulcer was adherent to the liver. In medical evidence[1] it was stated that the muscular effort used in shovelling gravel "would tend to increase the distance between the stomach and the liver," and that "there is always a tendency for the stomach to draw away from the liver." Since the ulcer anchored the stomach to the liver, it is obvious that the stress set up would tend to tear the stomach away from the liver. That is, it would if the statement made had any basis in fact. The view practically presumes that the stomach thus adherent to the liver at one point is attached equally firmly to something else, for the third law of motion makes action and reaction equal and opposite. Then between these points there is a tug when shovelling movements are made. This implies obviously that the two points do not move correspondingly when the muscular effort is made. The argument set up implies, and must imply, that under normal circumstances the stomach moves with the part of the body to which its other end or part is attached, and not with the liver. It implies equally inevitably, that the liver does not move as one piece with the rest of the body, at least not to such an extent as to prevent an adhesion between stomach and liver from being tugged on when the body moves the other part of the stomach.

But is this true? Is it a fact that the liver does not move as one piece with the body? The onus of proof lies on those who tell us this new story. Notwithstanding the onus, it was assumed as a fact, although it is in direct contradiction to the most explicit teaching of anatomical authorities, as, for example, in Cunningham's "Anatomy." In anatomy books it is carefully pointed out that the liver is firmly held in position and cannot leave it. The main portion, the right lobe, is held in place by the forward projection of the vertebral column, which prevents any tendency to shift to the left. The upper part of the liver fits above into the double cupola of the diaphragm, of which the right cupola is higher than the left, preventing again any tendency to shift to the left. It is held up in the hollow of the diaphragm by atmospheric pressure, just as the head of the humerus is held in the glenoid cavity, and it cannot separate without the production of a vacuum between. But besides the atmospheric pressure there is the intraabdominal hydrostatic pressure reinforcing it. The abdominal walls hold up the hollow viscera, which, in turn, press upwards and assist in holding the liver against the diaphragm. One of the chief organs helping to support the liver in this way is the stomach, and the under part of the left lobe of the liver is actually marked by a permanent indentation where it is supported by the upward pressure of the stomach. This indentation is regularly described in the text books, and its meaning explained. This pressure will, of course, be greater when intraabdominal pressure is greater,

and less when it is less, but it is always a positive pressure, a push, not a "tendency for the stomach to draw away from the liver" as the court was told in that notorious case. An ulcer of the stomach occurring in the part of the stomach normally in contact with the liver would be likely to become adherent there, but increased intraabdominal pressure would be protective against rupture, not provocative. It is not in accordance with reason to find any "aggravation" from muscular effort, which increases intraabdominal pressure.

But during muscular effort the abdominal wall contracts and raises the intraabdominal hydrostatic pressure. The pressure within the stomach rises. Why, therefore, does it not rupture the ulcer? The answer is that the pressure on the outside of the stomach is always raised to exactly the same degree as the pressure inside. This is an inevitable physical fact. Boyle's law is not a plaything for man, and may not be pushed aside as incomprehensible, and seemingly contrary to common sense. It does not matter what anyone thinks or wishes or ordains, the inevitable remains the inevitable, facts remain facts, and truth remains truth.

The case of *Cleary v. Wilson Granite and Gravel Company* has served as a text on which to make a disquisition about perforation of gastric ulcer, and has perhaps made it more interesting than the more arid matter preceding it. I do not propose to say more about the case or about gastric ulcer except a few final remarks. I may be asked why it perforated, after all. Well, the real cause, a perfectly ample and satisfying cause for the perforation was supplied, but then submerged again by a witness who made a first statement to respondent's solicitor, and, later on, a second statement to a representative of the applicant. In the first statement the witness said the deceased left to get a drink, and that it was after this that he first complained. The second statement dropped this part out, and in evidence[1] the witness either denied or stated that he did not remember what was contained in the first statement, and suggested that he was then intoxicated.

Please note that the date was December 31, 1928, the time near noon, and it was right in the middle of the hot weather.

### Other Conditions.

There are numerous other conditions which offer material for discussion under the title of this paper, but I have already taken up so much time that no further discussion is possible here. But I will mention a few.

1. Hernia. Ordinary hernias depend on the effect of intraabdominal hydrostatic pressure on weak spots in the abdominal wall. But internal hernias belong to a different category.

2. Diabetic coma in a diabetic may be precipitated by an accident or by an operation.

3. Acute pneumonia is not infrequent in cases of fractured rib.

4. Acute gout has many times in my experience developed, perhaps for the first time, in people laid up with a fracture. At the present time I have in hospital a man recovering from cholecystectomy who developed a very severe attack of acute gout three days after operation.

5. I have seen a case of varicocele undergoing a process of natural cure by simple phlebitis diagnosed as torsion of the spermatic cord and certified as due to a man's work. One of my friends recently told me of what I am convinced must have been a similar case in which a hospital surgeon demonstrated it to his class as torsion of the cord.

6. Arterio-venous aneurysm of the cavernous sinus is usually the result of a head injury involving the base of the skull. I have seen two with this history, and one without.

7. Traumatic epilepsy should be mentioned.

8. Dissecting aneurysm has been attributed to the strain of a severe muscular effort.

9. Acute dilatation of the stomach is said to have occurred as the result of a blow on the abdomen. I have not seen it.

### Reference.

(1) 1929, Workers' Compensation Reports, New South Wales, Volume III, page 145. *Cleary v. Wilson Granite and Gravel Co., Ltd.*

## WHAT MAKES A GASTRIC OR DUODENAL ULCER PERFORATE, AND WHAT PROTECTS IT?

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THE title of this paper puts a question which many of us have asked ourselves, and which it is well to study before answering. I do not mean that it is very difficult, but people jump to conclusions rather than reason to conclusions, and jump wrong.

Let us here examine the fundamental problem, which is a physical one, taking necessary preliminary facts from a trustworthy authority. The authority taken for our guide here is Watson's "Intermediate Physics," an accepted university text book. The introductory material in this paper is taken from Watson's book, unnecessary parts or words being omitted. Having done this, I proceed to study the special problem with the aid of some diagrams which I have drawn to make the application of the facts clearer.

### The Physical Problem.

When a system of forces acts on a portion of matter, although they may not produce any motion of the body as a whole, yet they may produce a displacement of the various particles of the body relatively one to the other. Such a relative motion of the different portions of a body is called a strain.

When a body is strained by the application of external forces it in general resists the strain, forces being called into play by the relative displacements of its parts tending to cause it to return to its unstrained condition. This restoring force, called into play owing to the strain, is called a stress.

A body which offers a stress, tending to restore the body to its original condition when it is strained, is said to be elastic or to possess elasticity.

The elasticity of a body is measured by the ratio of the stress corresponding to a given strain to that strain, or:

$$\text{Elasticity} = \frac{\text{Stress}}{\text{Strain}}$$

There are two distinct types of strain, namely, that in which the volume is altered without change of shape, and the other in which the shape is altered without change in volume, and to which a special name, a shear, is given. Strains, however, often consist of a combination of the two types, so that both the volume and the shape are altered.

In the case of a volume change the strain is measured by the change in volume produced per unit volume.

In the case of a pure volume strain, the force perpendicular to the surface acting on each unit of surface must be everywhere the same in magnitude, and this force per unit area measures the stress. This constitutes what is called a hydrostatic pressure.

The only kind of elasticity of which a gas is capable is elasticity of volume or bulk elasticity, since it is only to a change of volume that a gas offers any permanent resistance. If the pressure acting on a volume $V$ of a gas is increased from $P$ to $P + p$, and as a result the volume becomes reduced to $V - v$, then the strain or deformation produced in a volume $V$ is $v$, and therefore the strain produced per unit volume is $\frac{v}{V}$ while the stress corresponding to this strain is $p$. Hence, since the elasticity of a body is the ratio of the stress to the strain it produces, the elasticity $e$ of the given gas is

$$e = p \div \frac{v}{V} = \frac{pV}{v}$$

Robert Boyle obtained a series of values of the volume of a given mass of air under different pressures, and as a result he was led to enunciate a law, which is known as Boyle's law, that the product of the volume of a given mass of a gas into the pressure is a constant for all pressures.

If $V$ is the volume of a given mass of gas, and $P$ is the pressure to which it is subjected, then Boyle's law states that so long as the temperature remains constant

$$PV = \text{constant}$$

If the pressure acting on a gas is increased from $P$ to $P + p$, and as a consequence the volume changes from $V$ to $V - v$, then, if the temperature is kept constant, according to Boyle's law we have

$$\begin{aligned} PV &= (P + p)\ (V - v) \\ &= PV + pV - vP - pv \end{aligned}$$

$$\frac{pV}{v} = P + p$$

But $\frac{pV}{v}$ is the elasticity of the gas. Hence we see that the elasticity is numerically equal to the final pressure. If $p$ is so small that we may neglect it compared to $P$, that is, if we measure the elasticity for a small range of pressure, we may say that the elasticity of a gas at a pressure $P$, and at constant temperature, is numerically equal to $P$, and is thus the same for all gases at this pressure. Since the pressure of the atmosphere is about $10^6$ dynes per square centimetre, the elasticity of a gas at this pressure is $10^6$ dynes per square centimetre.

The above is an epitome of the facts governing the behaviour of gases so situated as to be subject to stress. Let us now proceed to their practical application to the problem in a hollow viscus, and the results in the presence of a weak spot such as an ulcer.

### Muscular Contraction.

For the purposes of this study, the abdomen may be considered as a closed vessel, the walls of which are mainly muscular and in a condition of tonus. The abdominal wall is not flaccid, and by its muscular contraction the cavity is able to vary in volume. Although maintained in a state of relative rigidity by tonus, the degree of tension between the bony attachments of the muscle will vary with circumstances. During strong muscular effort the volume of the abdominal cavity is diminished and the tension of its muscular wall is maintained at its maximum, making it practically rigid, and during such periods the intraabdominal hydrostatic pressure rises. Various organs are contained within the abdomen, but the gastro-intestinal tract is the only part that concerns us here. In this inquiry it makes no essential difference whether the tract is a continuously open canal, or whether it is divided into compartments. The walls of the stomach and intestines are furnished with a muscular tunic, the viscus thus being enabled to contract or expand so as to vary the volume, and so also the hydrostatic pressure, within it. The only sort of rigidity possessed by the gastro-intestinal tract is that developed by the hydrostatic pressure of its contents against the wall of the viscus.

Let us pause here to review the situation hydrostatically. First of all there is the atmospheric pressure $P$ in which the body carries out its existence, a pressure measurable as dynes per square centimetre, as grammes weight per square centimetre, as pounds per square inch, or otherwise. This pressure $P$ is present within and without the walls of every hollow viscus. Secondly, we can get within the abdomen an additional pressure $p_1$, caused by contraction of the muscle of the abdominal wall. This pressure $p_1$, obeying the same physical laws, must be present within and without the walls of any hollow viscus enclosed in the abdominal cavity, exactly as the atmospheric pressure is. Thirdly, within an individual hollow viscus, for example the stomach, there can be a further pressure $p_{11}$, caused by contraction of the muscular wall of the viscus itself.

Then, when the atmospheric pressure is $P$, the

intraabdominal pressure is $P + p_1$, and the intragastric pressure is $P + p_1 + p_{11}$.

Let us now study the conditions with the aid of diagrams.

Diagram I is constructed to illustrate the simplest condition, when we make the gastro-intestinal tract one continuous tube, without any sphincters locking it off into separated compartments. The outer envelope A represents the muscular abdominal wall. The inner envelope B represents the muscular tunic of the stomach and intestine. Between the two is a space representing the peritoneal sac, though, of course, it is not really a space. The part G represents the site of the stomach, but which in the diagram forms a cavity continuous with H, which represents the rest of the gastro-intestinal tract. GH is, therefore, one cavity. At one part W is an infolding producing an incomplete wall between the stomach G and the rest of the tract H.

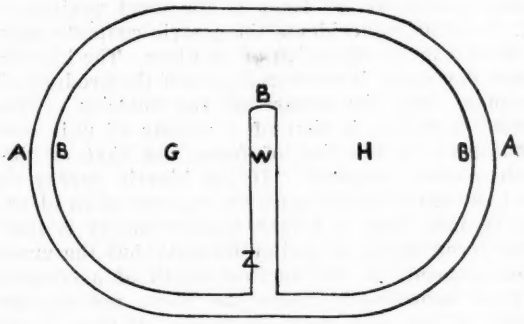


DIAGRAM I.

Outside of A, the only pressure is that of the atmosphere P, and it is supported by the same pressure within, but it is not necessarily the only pressure within the cavity GH.

Seeing that $PV$ = constant, according to Boyle's law the pressure must vary inversely as the volume. Therefore, in Diagram I, if the muscular wall A contracts, the volume diminishes and the pressure within rises in inverse proportion. Then, if the volume is reduced by $v_1$ to $V - v_1$, the pressure within GH rises by $p_1$ to $P + p_1$. Now the pressure $P + p_1$ is the hydrostatic pressure within the whole of the abdominal cavity, that is, the space enclosed by A. It is the pressure existing in every part of GH, and is measured as the pressure per square centimetre or per square inch, whichever way we like to put it, on the walls B of the cavity.

But if, while the part around H remains as it was, the part around G contracts so as to make the volume in G smaller again by $v_{11}$, the volume of the whole cavity GH is made smaller by $v_{11}$, and the hydrostatic pressure of the whole cavity GH is raised by $p_{11}$, not that of the G portion only. The same result would occur if the part around H contracted instead of that around G, and similarly for any contraction at any part.

Let us now suppose that in the partition wall W there is a weak spot, such as an ulcer Z, situated on the G aspect of it. Let us suppose further that the bursting strain of that ulcer is $p_{11}$. If $p_{11}$ is added to the existing hydrostatic pressure in G, it forms an addition to the total pressure in GH and is the same per unit area over the whole of the enclosing wall, and must be the same on both sides of W. Therefore, no strain can be produced at the ulcer, and it cannot burst.

The next case to consider is one in which, by a sphincter or other sufficient means the cavity G is made discontinuous with H. Diagram II has been constructed to illustrate this. It is the same as Diagram I, except that W now makes a complete division between G and H. For simplicity the diagram shows a system containing only two cavities, but it might with equal validity be a system holding any number of cavities.

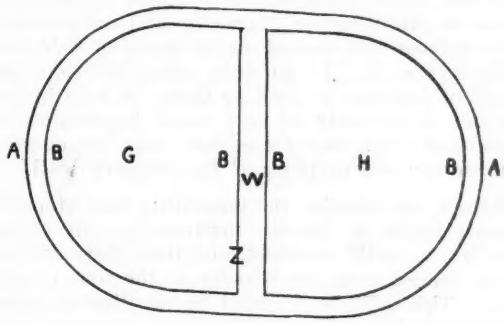


DIAGRAM II.

There are two sets of conditions to consider, the first is one in which the muscular tunic of the cavities is neutral, the second is one in which the muscular tissue of one cavity is active while it is inactive, or relatively inactive, in the other cavity or cavities. We can consider these one after the other.

If the muscular abdominal wall A contracts as a whole, or at any part, so that the volume of the abdominal cavity is reduced by $v_1$ to $V - v_1$, then the pressure within rises by $p_1$ to $P + p_1$. It is $P + p_1$ everywhere within the limits of A, so it is $P + p_1$ in G, and $P + p_1$ in H, and accordingly $P + p_1$ on either side of W. Therefore no strain can be produced at an ulcer on the partition wall. Nor can a strain be produced at the ulcer by any further increase of the general intraabdominal pressure.

But if now the muscular tunic B surrounding G contracts, it will add to the preexisting pressure in G, but in H the pressure will remain as it was before. Let B contract so as to reduce the volume in G by $v_{11}$ to $V - v_1 - v_{11}$. Then the pressure in G is raised by $p_{11}$, so that the total pressure becomes $P + p_1 + P_{11}$, while in H it still remains at $P + p_1$. A difference of pressure on the two sides of W is thus produced, tending to cause a strain. If the bursting strain of the ulcer Z is $p_{11}$, the ulcer will perforate as soon as the difference of pressure passes the threshold of $p_{11}$.

### Other Ways of Causing Perforation.

One can conjecture other ways by which a gastric ulcer could be caused to burst by pressure—pressure not due to contraction of the muscular tissue of the organ. They can be regarded as theoretically possible ways, but probably of no practical importance, for if they were to occur, they would in all probability be associated with other immediately fatal injuries, except in the case considered in the next paragraph.

When food and drink are taken into the stomach, the material, which will be practically liquid, will collect, under the action of gravity, at the most dependent part of the organ, and will remain there until forced on by the peristaltic contractions of the muscular tunic. This part is the greater curvature of the stomach, which is not a usual site for an ulcer. The mass of the stomach contents is subject to the influence of gravity, and the pressure at the bottom will depend on the depth of fluid vertically above it. It must be admitted that the "head" of pressure of two, or three, or four inches of water is probably of very small importance in comparison with the force that may be exerted by muscular contractions of the stomach wall.

But one can imagine the possibility that stomach contents might be thrown towards the site of an ulcer in a collision accident, and then there will be kinetic energy from the velocity at the time of collision. This will be directed in the line of force existing at the collision. The kinetic energy will depend on the value of $\frac{1}{2}mv^2$, where $m$ is the mass and $v$ the velocity. But $m$ is not the total mass of the gastric contents; the mass represented by $m$ is measured by the length of the column of fluid of unit sectional area traversed by the line of force. This may be a very different thing from the total mass or total weight of the gastric contents. There is, however, a possible additional factor which may have a qualifying influence. For if the rest of the mass of the contents is thrown against the gastric wall in the vicinity of the ulcer, it might produce some tendency towards stretching, so that tension is made on the ulcer. It is a factor difficult to evaluate. The total weight will not control the effect any more when the kinetic energy is developed by a fall from a height. When it is due to a fall from a height, the kinetic energy will depend on the distance fallen, which (with some qualifications) controls the velocity, and on the depth of fluid immediately vertical to the ulcer, so it is not every fall that can have an effect. There might not be any vertical depth of fluid actually above the ulcer. The body must drop so that when it is arrested a straight line between the surface of the fluid and the centre of the earth would pass through the ulcer. In all these cases the amount of destructive work that can be done on the ulcer in this way will depend on the value of $\frac{1}{2}mv^2$. But as in the case of collision accidents, there is a possible additional factor with a qualifying influence. This arises if a further part of the mass of the gastric contents is arrested by the gastric wall adjacent so as to stretch it and produce tension on the ulcer.

### Summary.

The physical stresses to which a gastric or duodenal ulcer may be exposed are reviewed. It is shown that by the operation of Boyle's law no increase of general intraabdominal pressure can cause a strain in an ulcer, and perforation cannot occur from that cause. It is shown that contraction of the muscular walls of the stomach will increase the hydrostatic pressure within that organ without increasing the general intraabdominal pressure, and if the difference in pressure passes the bursting strain of an ulcer, perforation must occur.

There are some other theoretically possible modes of causing perforation by pressure.

1. The weight of a column of gastric contents resting on the surface of an ulcer might conceivedly rupture it. Such an ulcer would only be on the greater curvature, at least in the erect position.
2. In collision accidents the gastric contents may be thrown in the direction of an ulcer. The kinetic energy developed is measured as half the product of the mass into the square of the velocity. The "mass," however, is that of a column of unit sectional area in the line of force, not that of the whole gastric contents. If the kinetic energy is great enough, it could cause the rupture of an ulcer.
3. In falls from a height, kinetic energy is liberated according to a similar formula, but the mass factor depends on the vertical depth of a column of fluid immediately above the ulcer, not on the weight of the total gastric contents. If there is no fluid vertically above the ulcer, this mass factor does not exist. With sufficient kinetic energy liberated to do the work, an ulcer could be ruptured.

In the three last cases there is a possible additional factor which may have a qualifying influence, for more or less of the rest of the gastric contents might lie, or be thrown, or drop on the adjacent gastric wall so as to stretch it, and thereby exert tension on the ulcer.

Cases 2 and 3 are hardly of clinical importance, since they would probably be associated with other immediately fatal injuries.

## SECONDARY CARCINOMA OF BONE.[1]

By L. J. CLENDINNEN, M.B., B.S. (Melbourne),
*Honorary Radiotherapeutist, Melbourne Hospital, Melbourne.*

IT is with pleasure I open the discussion on the treatment and results of treatment of secondary carcinoma of bone, for either the value of radiation is not sufficiently known or radiologists have failed to make any impression by recording their results. When it is realized that secondary carcinoma of bone is a manifestation of a general spread of malig-

[1] Read at the Second Cancer Conference, Canberra, March, 1931.

nant cells, whether it be by the lymph or blood stream, one cannot expect to read of percentage of patients alive after five years. So when a particular area of definite absorption of bone is treated, resulting in the disappearance of the patient's symptoms, followed by regrowth of the destroyed bone, then credit must be given to the treatment, even if the patient dies later of internal or generalized metastases. Recurrence is rare in an area that has regenerated after treatment, and some of these patients have been watched for some years by periodic X ray filming. It is not uncommon for patients to be relieved of their persistent and agonizing pain a few days after the first dose given, and this alone compensates for the many heartbreaks that occur when your patient, who is bedridden and comes to you in an ambulance, later walks normally in to see you and still later on dies of metastases of liver or lungs.

Secondary carcinoma of bone is most commonly seen in breast carcinoma, and is located generally about the sacro-iliac joints, sacrum, lumbar vertebræ, upper part of the femur and remainder of pelvis, in that order. The type is generally osteoclastic, and although the osteoblastic may occur or coexist, the latter type is much less likely to cause symptoms, and it is quite common in prostatic carcinoma to find a widespread involvement accidentally by routine X ray examination. In this disease, when the osteoclastic type is found, it is generally a terminal condition, and the results from treatment in my experience are not good, my only good result being in a man of eighteen stone, who was relieved of his pain for about fifteen months, to die of metastases elsewhere. As a rule these patients are in a markedly cachectic state, and do not respond, although the pain of about 50% of them is relieved.

The incidence of bone metastases from breast carcinoma is probably very much greater than is generally believed. According to *post mortem* records, the incidence varies from 20% to 75%, and is probably proportional to the thoroughness with which the examinations have been made. In the past ten years, at the Melbourne Hospital, one hundred and twenty cases have been diagnosed in the X ray department, and when I tell you only 35% of these were referred for treatment, you may realize that the value of deep therapy is not generally known or believed.

In order that you may believe me, I shall show you a few slides illustrating the results of treatment. The treatment in each case has been by means of X rays filtered through 0·5 millimetre of copper and four millimetres of aluminium. A full skin dose is given over a large field, generally anteriorly and posteriorly. This is followed usually by a second course of about 75% of the initial dose two to three months later, according to symptoms and condition of the patient's skin. In all, fifteen patients have been treated privately and forty-five at the Melbourne Hospital during the past nine years.

### Case Histories.

Case I is that of Mrs. C.P., aged thirty-seven, who was seen in January, 1925. She was suffering from carcinoma of the breast and had had an operation eighteen months previously. She had pain in the left leg for seven months with gradual loss of power. Figure I shows absorption of the lateral two-thirds of the left ilium. Figure II, taken two months later, shows regeneration of bone. A recurrence occurred in this case nine months later.

Case II is that of Mrs. J.S., aged seventy-three, who was seen in November, 1927. She had undergone an operation for advanced carcinoma of the breast two years before. She complained of increasing pain in the left hip for twelve months. Figure III is a skiagram showing patchy absorption of the supraacetabular portion of the left ilium.

Figure IV is from the same case two months later, showing regenerated bone in spite of a pathological fracture from a fall. This healing persisted, and the patient walked normally, but died nine months later from bronchitis

Case III is that of Miss M.C., aged forty-two years. X ray treatment was first given to her in December, 1926, for recurrent carcinoma of the breast. Pain developed in the right hip, with loss of power, and an X ray examination in November, 1927 (Figure V), revealed an absorption in the right ilium, in the superior mesial portions of the acetabulum, with a second area about the anterior spines of the ilium.

Figure VI is from the same patient two months later, showing regeneration in both areas. Sixteen months later this patient developed another focus in the left ilium, outside the area of radiation, which also responded to treatment. This patient died of an empyema eight months later, that is, two years after the commencement of treatment of the bone condition.

Case IV is that of Mrs. M.S., aged forty-eight years, who was seen in July, 1922. She had a right breast carcinoma removed two years before. Soon afterwards pain developed in the left shoulder, with loss of power for the previous fifteen months. A skiagram (Figure VII) revealed absorption of the upper lateral part of the body of the scapula involving the spine and upper portion of the glenoid. Figure VIII is from the same patient three months later, showing regeneration of bone and a recent fracture through the spine, which united, with further improvement in bony structure. The patient later was able to carry four gallons of water with this arm, and remained well for twenty-one months, when she died of pneumonia.

### Summary.

Secondary carcinoma of bone, which generally involves the pelvis and lumbo-sacral spine in breast carcinoma, responds to deep X ray treatment.

Almost every patient gets immediate relief from the pain, and a regrowth of bone frequently follows.

The pain in untreated patients who linger on, gradually increases until it becomes continuous and intense, and the relief of this by X ray treatment is a very real boon. Notwithstanding the fact that they usually die later on from generalized or internal secondaries, death then is generally painless and the result of asthenia.

The value of treatment of this type of local manifestation of a general malignant dissemination must be judged by the prolongation of life, accompanied by a change in the patient from a crippled condition, with constant pain relieved only by morphine, with the associated ill-effects, to one of comparative comfort, often compatible with the leading of a useful life.

# Reports of Cases.

## PERFORATION OF ŒSOPHAGUS WITH PULMONARY COMPLICATIONS.[1]

By GARNET HALLORAN, B.Sc., M.D. (Sydney), F.R.A.C.S.,
*Surgeon, Ear, Nose and Throat Department, Royal Prince Alfred Hospital, Sydney.*

R.S., A MALE, aged thirty years, a farrier, was admitted to the Royal Prince Alfred Hospital on March 5, 1930, with a history of having swallowed a fish bone four days previously. On admission he complained of dysphagia and severe pain to the right of the sternum and medial to the right scapula. His temperature was 38·9° C. (102° F.), his pulse rate was 134, and his respirations were 24 in the minute. He was obviously very ill, but showed no respiratory distress.

Dr. J. B. Langdon's X ray examination, prior to admission, revealed definite obstruction to the passage of a bolus at the level of the arch of the aorta, with some globules of barium outside the lumen. Dr. Langdon regarded the appearance as almost certainly due to a foreign body perforating the posterior wall of the œsophagus at the level of the aortic arch.

The patient's chest was carefully examined prior to operation, but no abnormal physical signs were found. On March 5 direct œsophagoscopy was performed under ether anæsthesia and a large fish bone was removed from an œdematous bed in the œsophageal mucosa. The extremely sharp spine of the foreign body, seen in Figure I, was pointing into the patient's right postero-lateral œsophageal wall, and in view of the patient's bad general condition and the X ray report, this was considered to be the site of a possible œsophageal perforation. A provisional diagnosis of septic mediastinitis was made.

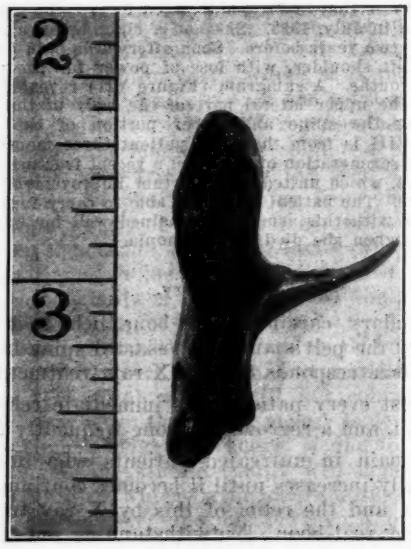


FIGURE I.
Showing foreign body of which the sharp spine had impaled the œsophageal wall.

The patient had no further symptoms referable to the œsophagus, but on the day following operation he complained of pain on the right side of the chest. His respirations had increased in rate from twenty-one to forty per minute, the pulse from 100 to 124 per minute, and the temperature, which had fallen to normal after operation, rose again to 37·8° C. (100° F.).

Dr. C. G. McDonald, who saw the patient at my request on March 7, found abnormal physical signs at the base of the right lung, namely, impaired note on percussion (but not absolute dulness) and absence of breath sounds. At this time the respirations were rather hurried and shallow.

The patient was transferred to his care. The next day (March 8) the chest was examined by X rays and the following report obtained: "No evidence of fluid in the thorax. The right hilum is increased and there is considerable infiltration in the right lower lobe. The appearance is probably due to mediastinitis."

On March 17 a white blood cell count revealed 16,750 leucocytes in each cubic millimetre of blood. Of these 80% were neutrophile cells, 14% were lymphocytes, 5% were monocytes and 1% eosinophile cells.

On March 20 examination of the sputum failed to reveal the presence of Koch's bacillus. The predominating organism was a pneumococcus. Streptococci, staphylococci and *Micrococcus catarrhalis* were also present. The sputum was profuse, being purulent for several weeks.

On March 20 the radiologist reported marked dulness towards the base of the right lung, especially the cardiophrenic angle; the appearance suggested a bronchiectatic condition in this region; the left lung appeared clear. Dulness to the right of the mediastinum was slightly less pronounced than in the previous examination (see Figure II in the Supplement).

For a period of five weeks the patient's temperature chart showed a sharp evening rise between 36° and 39·6° C. (100·5 and 103·4 F.) and a morning fall to normal or to slightly below the normal line. At the end of this period he remained afebrile. On April 17 all pulmonary signs and symptoms had cleared up and the patient was convalescing.

I have discussed the pulmonary complications of this patient with Dr. McDonald. He agrees that a consideration of the physical signs and the X ray shadows pointed to an infection of the right half of the lower mediastinum, with pneumonic consolidation of the right lower lobe. Whether the latter resulted from the former, however, he considers doubtful, owing to anatomical difficulties. The most probable explanation is that an infection of low virulence formed, by extension from the œsophagus, in the right lower mediastinum, and the pneumonic process in the lower pulmonary lobe was consequent on the anæsthesia. It is just possible that a low grade pyogenic infection of the mediastinum ruptured into a bronchus and set up a bronchopneumonic inflammation of the right lower lobe from which the patient fortunately recovered.

[1] The patient described herein was shown at a meeting of the New South Wales Branch of the British Medical Association on May 14, 1931.

# Reviews.

## PRIMARY SYPHILITIC LESIONS IN WOMEN.

DURING the war and in the years following it a considerable literature has been published dealing with early syphilis in the male. The study of the earliest manifestations in the female has not received a similar attention, and in "Primary Syphilis in the Female" the author has supplied an illuminating account of his observations in this field.[1] He points out that it is generally believed that the primary syphilitic lesion is uncommon on the internal genitalia of the female. His statistics of 384 primary genital chancres in women reveal that 45% were internally situated. It is clear that this large percentage would remain undiscovered but for a careful internal medical examination. Again, he shows that the commonest site of infection is on the cervix, for in his series no fewer than 44% favoured this situation.

The reader will find in this work a great deal of useful information founded on the author's experience. It is excellently illustrated by colour photographs as well as untouched photographs prepared by the author himself.

No one who is called on to diagnose primary lesions in the female, would wish to be without this carefully compiled and illuminating account of this aspect of syphilis.

[1] "Primary Syphilis in the Female," by T. A. Davies, M.D.; 1931. London: Humphrey Milford; Sydney: Angus and Robertson. Demy 8vo., pp. 119, with illustrations.

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## CONTRACEPTION AND CANCER.

No man in his right senses would say that every possible avenue in the causation of malignant disease should not be investigated. Anyone who held such a view would be treated with scorn and derision, if not with pity. But all avenues are not equally pregnant with possibilities; some are barren. Every investigator will agree that trauma may play some part in the causation of cancer—trauma either in the form of direct injury or in the form of chronic irritation from excessive function, from the presence of chronic disease or from external agents. When a source of chronic irritation is known to exist, it can be removed, but hypothesis alone is not sufficient ground for drastic steps intended to be prophylactic.

At the Second Cancer Conference held at Canberra a few months ago a resolution was adopted recommending to each State organization the desirability of studying the effects of contraceptive agents in relation to uterine cancer, and suggesting that reports should be prepared for consideration at the next conference. Motions are easily worded and their passing as resolutions is quite simple. This resolution at first sounds reasonable and the projected course of action desirable.

Since contraceptive agents have no direct action on the body of the uterus, cancer of the cervix alone need be considered in the present discussion. Cancer of the cervix is almost exclusively a disease of parous, generally multiparous, women, and not of those who have borne no children. Multiparous women include those who use no contraceptive agents at all as well as those, of course, whose contraceptive agents have failed them. Nulliparous women include both those who have had no sexual intercourse and those (probably an exceedingly large group) who have found contraceptive agents or methods effective in their sexual relationships. It would be most astounding if contraceptive agents, acting as a cause of cancer, had a selective action on the cervices of women who had borne children. That trauma associated with parturition is an ætiological factor in cervical cancer of the parous woman is a justifiable conclusion.

If an endeavour were to be made to prepare a report on the use of contraceptive agents in relation to cancer, it would be difficult to know how to set about it. The investigation would have to be most extensive. It would be necessary not only to ascertain the history of every woman with uterine cancer, particularly in regard to her use of contraceptives, but, in addition, large numbers of non-cancerous women, nulliparous and parous, would have to be interrogated and examined. The difficulty of obtaining what might be called the contraceptive history of parous women would be great; it would be nothing compared with the task of obtaining a similar history from nulliparous women. When the histories were obtained the percentage of error from deliberate deceit would be enormous. The task suggested by the Cancer Conference would be particularly difficult, for it is suggested that attention should be directed to contraceptive agents; methods are excluded. Such practices as *coitus interruptus* would therefore not be considered. Attention would be devoted especially to the use of suppositories and such appliances as cervical caps; presumably the use of condoms would be included. Coal tar derivatives, such as those used in weak solutions for purposes of syringeing, would be included. When the

history of parous women was obtained in such an investigation it would be necessary to place in a separate group or groups those who had cervical lacerations, and the difficulty would arise of defining the degree of trauma sufficiently extensive to warrant separate grouping.

The impossibility of obtaining information from which reports might be drafted for the next cancer conference is obvious. Such an investigation would have to be spread over all the States of the Commonwealth, and several years would necessarily elapse before sufficient data were accumulated. If an investigation were undertaken, information should be sought particularly in regard to the use of chemical contraceptives by patients with cervical cancer. Contraceptive agents probably lie in one of the avenues already described as barren. There is no doubt that the main cause of cancer of the cervix is trauma at parturition. If one could bring about a state of obstetrics in which every child were born by Cæsarean section, it might be possible to assess the ætiological rôle of contraceptives in what would then be a small residue of cervical cancer. At present contraceptives might be regarded by some as preventive of cancer, since by preventing pregnancy they prevent parturition and its attendant cervical laceration. Energy should be expended in the prevention of cervical tears and on insistence on their immediate repair when they are not preventible. As far as contraceptives are concerned, the medical profession would do well to study the indications for their use, so that they may be prepared to give definite instructions to their patients when need arises.

## Current Comment.

### THE ASSOCIATION OF SYPHILIS WITH CANCER.

IN 1930, G. H. Belote applied the Wassermann or Kahn test in a series of ninety-two cases of cancer of the tongue; he found a positive reaction in 29·3%. Among all patients admitted to hospital there was a positive blood reaction in slightly under 6%. In all cases of cancer located elsewhere than in the tongue the blood reaction was positive in 7·2%. The suggestion was made that syphilis may sometimes be a factor in determining carcinoma; if a malignant tendency exist, recurrent syphilitic lesions may act as a precipitating factor. To ascertain if any constant relation existed between syphilis and carcinoma, as shown by reactions to blood tests, a study was made of 1,267 cancers in common sites.[1] Positive serological reactions were given in the following percentages in cancer in different sites: *Cervix uteri,* 15·1; vulva, 11·8; lip, 8·8 (distributed as 7·9 in males and 28·6 in females); skin, 5·3 (6·0 in males and 3·1 in females); stomach, 3·7 (3·4 in males and 5·3 in females); rectum, 4·5 in males and none in females, 3·5 in all; breast in females, 3·3. Of the 1,267 patients 6·6% gave a positive reaction; this is in contrast with 7·2% in Belote's previous study. Considering the greater number in the second series, the lower figure is probably more nearly correct, but the difference is not great. In the previous study the incidence of positive reactions in female patients was 2·4% greater than in males similarly afflicted. In the present study the difference is 2·7% (8% in females and 5·3% in males). Taking the average incidence of positive reactions as 6·6%, an incidence higher than the average was shown in malignant disease of the cervix, vulva and lip, and a lower than average incidence in cancer of the breast, skin, stomach and rectum. In a series of 58 cases of carcinoma of the penis and *fundus uteri,* not one gave a positive reaction. But diagnosis of cervical carcinoma is not always easy, and serious errors may be made if the peculiar changes accompanying epithelialization of the cervix after inflammatory lesions be not fully considered. Emil Novak emphasized these changes. L. C. Scheffey (1931) found a positive Wassermann reaction in 7·6% of his gynæcological patients, as compared with Belote's 8·0% in all carcinomatous females. Belote's patients with carcinoma of the cervix gave over 15% of positive Wassermann reactions, and carcinoma of the vulva nearly 12%. The 15·1% of positive reactions in cervical carcinoma is nearly three times the average for all hospital admissions and almost twice the average for all carcinomatous female patients. The number of cases of cancer of the vulva (seventeen) was too small to warrant definite conclusions. But F. J. Taussig (1929), in a series of 76 vulvar carcinomata, observed that eight developed on a gummatous basis, and in these the average age was thirty-eight, as contrasted with sixty-four years where the malignant growth arose on a leucoplakic area. The conclusion was that if syphilis did not actually cause malignant degeneration it might be a factor in hastening it.

Cancer of the lip is much more common in males than in females. In this series the number of female patients (seven) was too small to admit of definite conclusions, but the discrepancy in percentage of positive Wassermann reactions (over twenty-eight in females and under eight in males) is remarkable. Patients with skin cancer show a percentage of positive blood reactions slightly lower than that of routine hospital registrations. It

[1] *The American Journal of Syphilis,* July, 1931.

would seem, therefore, that syphilis is not a potent factor in the production of cancer of the skin. In carcinoma of the breast, stomach and rectum the percentage of positive blood reactions was definitely below the average for all hospital registrations. In cancer of the *fundus uteri*, out of 35 patients not one gave a positive reaction. Each of these structures is associated with columnar epithelium, and none is commonly associated with recurrent syphilitic lesions. On the other hand, the organs which display a higher than average association of carcinoma with syphilis, have squamous epithelium and are commonly the sites of recurrent syphilitic lesions. These same structures are continuously exposed to repeated irritations and therefore predisposed to malignant degeneration. The integument in general also has squamous epithelium, but here many other factors are concerned, and though they may increase the actual numbers of carcinomata they probably lessen the percentage of association with syphilis. The explanation of complete absence of positive serological reactions in the twenty-three cases of penile cancer is not clear. Perhaps irritative factors which do not obtain elsewhere in the body, are concerned.

Belote does not infer that syphilis causes cancer, but he believes that recurrent lesions tending to interrupt surface epithelial continuity may be an additional factor in cancer production, especially in cancer of squamous epithelium. The unexpectedly high percentage of positive serological reactions in carcinoma of the *cervix uteri* suggests that this organ may be more often the site of syphilitic lesions than has been considered, and might be more often found if specially sought.

H. J. B. Fry[1] has compared the serum reactions in one thousand unselected patients with cancer in the Cancer Hospital, London, with those attending hospital for other ailments. Positive reactions in malignant conditions were noted in 9·7% and in the control cases in 13·2%. The percentage of positive reactions was 6·2 in 535 cases of malignant disease elsewhere than in the buccal cavity. In eighty cases of cancer of the tongue there was a positive reaction in 35%. In the seventy-six male patients the percentage was 36·8. Fry did not find so great a disproportion between the sexes regarding syphilis of the tongue as in cancer, and was of opinion that to account for the great incidence of cancer in syphilitic tongues some other factor, such as smoking, must be considered. Other percentages found by Fry were: Lips, 29·4%, all in males (compared with 21·4% recorded by Simmons and Daland); jaw, 20·8% (with a slight preponderance in males); combined bucco-labio-pharyngeal percentage, 25·4% (as compared with 35% found by Roussy and Bertillon and 16% by Simmons); *cervix uteri*, 13·3% (as compared with Martzloff's 6·8%). Fry's conclusions were that there is a lower percentage of positive Wassermann reactions in malignant disease than in non-malignant conditions. Excluding buccal conditions, he found that the percentage is only half of that in non-malignant lesions. Fry states that, excepting cancer of the stomach, there is a low percentage of syphilis in cancer of the alimentary tract, and the incidence diminishes from the mouth to the anus. Syphilis in his opinion is not an important factor in cancer of glandular organs nor (excluding the lip) in cancer of the skin. In general he found no evidence that syphilis plays any important or direct part in the production of cancer.

The statistics provided by Belote and Fry disclose remarkable discrepancies and their conclusions are totally at variance. To arrive at a just appreciation of the facts of any association of cancer with syphilis a very much larger number of cases must be considered. Statistics based on comparatively small numbers are liable to error and are not convincing. The present widespread practice of cigarette smoking among females may confuse the estimate of the part played by smoking as a factor in determining cancer of the lips and tongue in males. But it is not even now universally agreed as to what part, if any, smoking does play in predisposing to malignant disease.

[1] *Journal of Hygiene*, Volume XXIX, 1930, page 313.

## PALLIATIVE TREATMENT OF ŒSOPHAGEAL CANCER.

CANCER of the œsophagus is one of the conditions for which no satisfactory treatment has yet been devised. The performance of thoracotomy for the purpose of implanting radon needles has been reported and experimental work on the surgery of the œsophagus has been carried out. The work of J. H. Saint on the œsophagus of the dog was reviewed in these pages in December, 1929. Treatment has eventually to be purely palliative and as a rule gastrostomy or enterostomy is chosen. D. W. Gordon Murray has reported a method of palliative treatment which should prove useful.[1] It is based on principles which, Murray states, were first laid down by Simmonds and later by Souttar. The aim is to dilate the stricture and to pass through its lumen a tube which will prevent closure by spasm or growth. Murray admits that there is danger of splitting the wall of the œsophagus and causing infection of the mediastinum; in his experience this has not occurred. The tubes used consist of coil spring wire, an alloy of silver and nickel. The tube is inserted and left *in situ*. Murray claims that the fine calibre of the wire makes a soft tube which does not produce ulceration. The metal tarnishes, but does not corrode. Murray gives the histories of ten patients who were treated by this method. All but one were able to return to their occupations. They were all able to take food naturally and lived in comparative comfort. The longest period during which the treatment was carried out was ten months. The method is worthy of careful trial.

[1] *The Canadian Medical Association Journal*, September, 1931.

# Abstracts from Current Medical Literature.

## PÆDIATRICS.

### Sarcomatosis.

EDOARDO PRETO (*L'ospedale Maggiore*, May, 1931) describes a rare case of sarcomatosis. A female child, apparently in excellent health, fell, striking her forehead. There was only a slight bruising of the injured region, but for the first two days the child was very restless. On the third day the mother noted a small, painful swelling in the right parieto-occipital region. The child became rapidly worse, the occipital tumour grew to the size of half an orange. Other nodules, from the size of a pea to that of a pigeon's egg, appeared in the parietal region, the maxillæ and the mandibles. Bilateral exophthalmos followed, not pulsating, and more marked on the left side. Twenty-four days after the fall the child died. An autopsy disclosed a sarcoma probably originating in the orbit, with metastases in the *dura mater*, both breasts, the pleura, both lungs, the mediastinum, liver, kidneys, bones of the cranial vault, ribs, spine and pelvis. The spleen and lymph glands were free. Histologically the growth was found to be a small round-celled sarcoma. It resembled somewhat a chloroma, but the characteristic green colour was lacking. The author believes that metastasization took place by the blood stream, the carried neoplastic emboli proliferating at a distance. The rôle of trauma is discussed. In the author's opinion the tumour was of congenital origin and the trauma acted as an exciting cause.

### Chronic Pulmonary Infections in Childhood.

ALLEN K. KRAUSE (*Tubercle*, May, 1931) discusses the importance of recognizing and treating at an early stage in children chronic focal pulmonary infection, either that due to the tubercle bacillus or that caused by other microorganisms. In regard to the former, he points out that in the period of childhood, contrary to older belief, there is extraordinarily high resistance to the tubercle bacillus. Tuberculosis in children is essentially a non-progressive infection. Though the opportunities for infection are here at a maximum, the mortality from tuberculosis is by contrast at a minimum during childhood. Krause insists on the need for recognizing that obscure ill health in a child may be due to tuberculous infection, and pleads for a more general use of tuberculin tests as aids to diagnosis. He scorns the term latent tuberculosis, maintaining that a child either has active tuberculosis, that is to say, infection with the tubercle bacillus of such degree as to merit medical attention, or there is no disturbance of function and the tuberculosis is inactive; he will admit to no middle ground. He deplores the fact that to most physicians tuberculosis has far too much usurped the field of attention to chronic pulmonary infections. Non-tuberculous focal infection of the lung in infants and children are both common and serious. They may retard development and lead to invalidism. Bronchiectasis in particular is diagnosed more often late than early, though here more than in tuberculosis early diagnosis is essential if treatment is to be of any avail. In many children there is to be found a similar clinical history of pulmonary infection in infancy followed by repeated colds or attacks of bronchitis, especially during winter. A permanent focus of infection in the lung is postulated, whence arise exacerbations of pulmonary infection, similar to those caused by the tubercle bacillus. Later clinical and radiological evidence of inflammation will be found. Eventually these focal infections may go on to bronchiectasis or abscess. Though upper respiratory sepsis has usually been found and treated in these children, the pulmonary infection remains. Pathologically, little is known of its exact nature until gross changes have occurred. The author finally urges that in view of our lack of therapeutic resource in dealing with the end results of these focal infections, fresh colds and exacerbations should be prevented as far as possible. Some form of sanatorium routine should be instituted, as, if a long enough period of quiescence can be obtained, the damaged area may become permanently healed.

### Metabolism Studies in Cœliac Disease.

O. MACRAE AND N. MORRIS (*Archives of Disease in Childhood*, April, 1931) have continued their work in cœliac disease, stimulated by Moncrieff and Payne's conclusion that the disease is chiefly due to an impaired action of blood on tissue lipase. Moncrieff and Payne found a high blood fat content in cœliac disease. The authors and Fanconi have not been able to confirm these results. They demonstrated that carbohydrate and acid sodium phosphate were also absorbed with difficulty, whereas urea, sodium chloride and methylene blue were absorbed with equal rapidity, perhaps due to the fact that they pass through the intestinal wall by simple diffusion. In a case of tuberculous peritonitis with steatorrhœa, carbohydrate and protein were absorbed quite normally. There is no evidence of any structural change in the intestine which could account for the grave defect in absorption, the mucosal atrophy being a result of starvation. In two cases in which a barium meal was observed, there was no abnormality in its times of transit; this does not support the theory of defective absorption from intestinal hurry. By exclusion, since intermediate metabolism, intestinal epithelium and rate of transit are all normal, the fault must lie in some physico-chemical abnormality of the intestinal contents. The authors suggest that a paucity of bile salts may explain the defective absorption. In two of their own cases the results were conflicting; one patient improved with bile salts administration, the other failed to improve. They point out that cœliac disease metabolism, however, differs from conditions of pure biliary atresia. The beneficial effect of acid sodium phosphate they attribute to its acidity rather than its phosphorus content, stimulating the production of secretion and bile and leading to a better mineral and fat absorption. If vitamin *D* is absorbed more readily with the fat, fixation of calcium and phosphorus in the bone would naturally follow.

### Nutritional Xerophthalmia and Congenital Night Blindness.

J. C. SPENCER (*Archives of Disease in Childhood*, February, 1931) has made a clinical study of nutritional xerophthalmia and congenital night blindness in an urban industrial population. He found that, though a large number of children may be living on the same deficient diet, only a few become affected. No relationship was noted between the general state of nutrition and the occurrence of the disorder; and cure by the appropriate vitamins was not accompanied by a coincident rise in height or weight. Further, there was no evidence of rickets or other deficiency disease in any of the patients affected, but a high incidence of skin sepsis was noted. Large numbers of epithelial cells occur in the urine, and this confirms the discovery of Wolbach and Howe that the essential and specific lesion in vitamin *A* deficiency is debasement and damage to epithelial structures. Irradiated ergosterol, ultra-violet rays and liver extract had no therapeutic effect, but constant cure followed the addition of only ten cubic centimetres of cod-liver oil to the daily diet.

### The Use of Respiratory Vaccine in Pneumonia.

ROY FORBES AND CHARLES STEINBERG (*Archives of Pediatrics*, April, 1931) refer to the excellent results of vaccine therapy in pneumonia reported by Wynn, Zinssner and others in adults. They have applied the principle to children, using Lambert's technique, in which large doses are used at frequent intervals, for example, every six hours until lysis occurs. A mixed concentrated vaccine was employed, containing 2,000 million bacteria to the cubic centimetre. Use of the vaccine must be commenced early, and patients in hospital are usually not "early cases." Twenty-six children were treated at home with no mortality, with good results in 75·9%. Fifty hospital patients received vaccine over a period of two years, as compared with sixty-six receiving routine therapy during the same period. The vaccine-treated patients usually showed an improvement as compared with the controls during the first twenty-four hours, and lysis occurred very consistently about the fifth to seventh day. The

distressing cough was not modified, however. A mortality of 18·7% was noted, as compared with that of 27·3% in the control series. When treatment was instituted within forty-eight hours of the onset the mortality was 4·6%, as compared with 20·6% in the controls. The results on the whole were strikingly similar to those obtained by Lambert and Sutton in adults.

### Salicylic Acid Fruit in the Prevention and Treatment of Rheumatism.

J. EPSTEIN (*Archives of Pediatrics*, February, 1931), as a result of a study of 131 children of all classes, is convinced that salicylated fruit juices are valuable as a prophylactic antirheumatic, and should be added to the diet of every child, especially those with an inherited susceptibility to the disease, from the second year of life onward. Fruits containing small quantities of salicylic acid in a natural active state are strawberries, huckleberries, plums, raspberries, cherries, lemons, grape-fruits and melons, so that a selection can be made to suit the child's individual taste. This administration should not replace a rich dietary, sunshine, avoidance of damp and fatigue, eradication of septic foci and the administration of quinine and calcium salts, which are the accepted means of combating the rheumatic complex.

## ORTHOPÆDIC SURGERY.

### Treatment of Chronic Osteomyelitis with the Maggot.

W. S. BAER (*The Journal of Bone and Joint Surgery*, July, 1931) states that maggots have been found to be a useful adjunct to thorough surgical treatment of chronic osteomyelitis and far more successful in securing permanent healing of these extensive wounds than any other method. Maggots, by their digestive action, clear away the minute fragments of bone and tissue sloughs caused by operative trauma in a way not accomplished by any other means. This is a valuable asset in the healing of a wound. Maggots cause wounds to become alkaline and in this way diminish growth of pathogenic bacteria. The question is under investigation as to whether maggots have more subtle biochemical effects within the wound itself and perhaps cause also a constitutional reaction inimical to bacterial growth. The eggs of the flies are immersed in a one in two thousand parts of bichloride of mercury, 25% alcohol and one-half of 1% hydrochloric acid for thirty minutes, which sterilizes them so that they may be used in any wound without risk to the patient. The author considers that the post-traumatic or post-operative general condition of the patient is better in maggot treatment than in the older forms of treatment, in which infection was combated by chemicals or other types of dressing. There is less absorption and less toxic reaction. In open tuberculous abscesses with or without secondary infection, wide exposure followed by maggot treatment has proved surprisingly effective in a small number of cases and will be given further trial.

### Muscular Relaxation in Tendon Repair.

R. W. MCNEALY AND MANUEL E. LICHTENSTEIN (*Surgery, Gynecology and Obstetrics*, July, 1931) discuss muscular relaxation produced by "Novocain" as an aid in tendon repair. Clinical experience led them to believe that retraction of the proximal end of the cut tendon was one of the most formidable obstacles to immediate suture and subsequent repair. Likewise, it was felt that the development of a method which would either diminish or abolish this retraction would serve to decrease the number of failures in tendon suture. It seemed evident that relief of tension on the suture line would favour union, permit of the use of less suture material, and lessen the likelihood of the tearing out of the sutures used in repair. "Novocain" injected intramuscularly definitely removes the influence of the nervous system on the muscle injected. In the unanæsthetized dog the duration of this elimination of nerve influence following a single injection lasts a variable period of time, from thirty minutes to four hours. Repeated injections with "Novocain" of a muscle the tendon of which has been severed, is sufficient to induce continuous muscular relaxation by eliminating the primary hypertonic contracture. The decrease of tendon retraction permits an earlier healing by decreasing the amount of fibrous tissue which is necessary to fill in the gap which is produced in the severed tendon.

### Breaking Strength of Healing Fractures.

M. K. LINDSAY AND E. L. HOWES (*The Journal of Bone and Joint Surgery*, July, 1931) describe a quantitative method indicating the breaking strength of regenerating bone. Strength manifested itself by the sixth day and then increased rapidly to the twenty-first day. It was noticed that the callus grew steadily larger during this time. A period of decline in strength occurred after the twenty-first day, during and following which time the callus decreased in size. A secondary rise in strength from the thirty-third to the forty-fifth day also occurred. Strength was attained on the twenty-first day, which is comparable to that on the thirty-ninth, but a period of weakness existed between the two. There was a direct correlation between the results of this study and previously observed histological facts and clinical data.

### Orr Treatment of Osteomyelitis and Allied Suppurative Processes.

JACOB KULOWSKI (*The Journal of Bone and Joint Surgery*, July, 1931) recommends the Orr method as a routine procedure in the treatment of osteomyelitis and allied conditions because of the following reasons: (i) It is based on the principle of adequate drainage and adequate immobilization; (ii) it is a painless, economic, universal method applicable in any stage of osteomyelitis; (iii) the period of stay in hospital is greatly decreased; (iv) transportation of the patient is simplified; (v) it prevents sequestration; (vi) the general condition of the patient is improved; (vii) a good functional end result is obtained; (viii) it minimizes loss of limb by amputation; (ix) the death rate is insignificant; (x) it is the only treatment that satisfies all the tenets of orthopædic surgery; and, lastly, it shortens the post-operative course of a condition which, previous to the advent of the Orr method, was in many cases intractable.

### Intracapsular Fracture of the Hip.

M. O. HENRY (*Journal of Bone and Joint Surgery*, July, 1931), after experimenting with Hey Groves's method of proximal osteosynthesis on dogs in fifteen instances, concludes that round nails are inadequate for fixation of the fragments of intracapsular osteotomy of the neck of the femur without external fixation. Satisfactory contact and adequate fixation of the fragments of intracapsular osteotomy of the neck of the femur in dogs were obtained by a rustless steel screw of a special pattern, which was practically inert in bone. The author considers that early functional stimulation of the circulation is of prime importance in the healing process of intracapsular fracture of the hip, and that early, continuous and accurate contact with absolute fixation of the fractured surfaces is of paramount importance in the healing of fractures in the presence of synovial fluid. Finally, the capital fragment, devoid of all soft parts, in intracapsular osteotomy of the neck of the femur of the dog readily unites and revivifies under the proper physiological conditions essential to the healing process in bone.

### Measurements of the Lower Extremities.

R. SOMMER (*Deutsche Medizinische Wochenschrift*, March 27, 1931) considers that the average text book gives incorrect instruction regarding the points from which to measure the lower limbs for differences in length in fractures, dislocations or other deformities. The usual advice is to measure from the anterior superior iliac spine to the external malleolus. This is liable to gross errors according to the amount of abduction of the limbs. He advocates placing the patient on a flat surface with the limbs parallel and marking four points—the internal malleolus, inner margin of the knee joint, trochanter and anterior superior spine. A comparison of corresponding points on each side will at once reveal any abnormality. The article is illustrated with diagrams of selected cases to prove the author's contentions.

# British Medical Association News.

## SCIENTIFIC.

A MEETING OF THE NEW SOUTH WALES BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the Robert H. Todd Assembly Hall, British Medical Association House, 135, Macquarie Street, Sydney, on July 30, 1931, the President, DR. GEORGE BELL, in the chair.

### Trauma and Visceral Organic Disease.

DR. S. A. SMITH read a paper entitled: "Trauma and the Heart" (see page 575).

DR. C. E. CORLETTE read a paper entitled: "Trauma in Relation to Organic Visceral Disease" (see page 583).

DR. JOHN STOREY prefaced his remarks by stating that he had not been concerned in the case *Cleary versus the Wilson Granite and Gravel Company, Limited*. He thanked Dr. Smith for his most interesting paper; it would appear that physicians did not always know so much about a given case of heart disease as they would have others believe, as with surgeons and their patients. He agreed with Dr. Corlette's remarks about perforating ulcer; there was no question that most of these ulcers perforated within a short time after the ingestion of something. In the case mentioned by Dr. Corlette it had probably been beer, as it often was in hospital cases. Speaking of work as an alleged cause of appendicitis, Dr. Storey had seen such an instance. A man was given a certificate that he had had appendicitis due to a certain accident. Dr. Storey could not understand this and had questioned the doctor who had given the certificate. It appeared that the certificate was dated before the operation for appendicitis and had been given during convalescence. There had been no blood in the abdominal wall, no ecchymosis in the peritoneum, the position of the appendix was retrocaecal. The tenet could not be sustained.

Dr. Storey wished to ask Dr. Corlette one question particularly: Supposing someone dear to himself had an ulcer such as had been described, would he advise that patient to exert himself laboriously if the ulcer was on the point of perforation, or would he advise the patient to keep still?

Dr. Storey said that he was the friend who had told Dr. Corlette about twisted pedicle of the testis. He had seen in London a youngster demonstrated with a distended testicle and some thrombosed veins, and had been told by the demonstrator that it was a case of twisted pedicle. The next morning, in another class, that of Jonathan Hutchinson, he had been asked to examine a patient suffering from an identical condition; it was another case of torsion of the testicle. Dr. Storey had not, however, seen another case since. He maintained that Dr. Corlette's explanation did not hold. They did not know what caused the condition; something had caused thrombosis; but this was not necessarily Nature's way of curing a varicocele.

DR. C. G. MCDONALD congratulated both speakers on the excellence of their respective papers. In regard to Dr. S. A. Smith's paper, he believed it impossible to criticize those portions which dealt so admirably with the action of effort on the healthy heart and with medico-legal cases under the caption of "cardio-neuroses." It was an established fact that it was impossible to produce failure of the healthy heart, notwithstanding lay (and sometimes medical) opinion to the contrary. The workers' compensation acts had given medical men the opportunity of seeing many patients suffering from alleged "heart strain," whose hearts on physical examination proved to be normal. Many persons had an expressed or secret fear of heart disease, and if they experienced pain over the precordium when performing some strenuous task, they became afraid that the heart had been injured. The majority of these patients were thoroughly sincere in their fears. Many of them complained of the same sensations—pain over the precordium, followed by such vasomotor symptoms as dizziness, faintness and momentary dimness of vision, and though they might not be breathless at first, they manifested either the effort syndrome or hysterical dyspnœa at a later stage. If the medical attendant made a diagnosis of "dilated heart" or "heart strain," the patient's fear became a conviction difficult to eradicate. If, however, the attendant recognized the functional nature of the condition and was able to explain the mechanism of the symptoms and to reassure his patient, rapid recovery followed.

But while Dr. McDonald agreed that it was impossible to damage the healthy heart by exertion, however intense or prolonged, there was one point in Dr. Smith's paper to which some gentle criticism might be applied. He knew Dr. Smith as a writer of charming papers which tended to be provocative; this paper was a challenge to the ignorance which existed in regard to the effect of effort on the diseased myocardium. There was little doubt, as Dr. Smith had maintained, that violent effort might hasten the onset of fibrillation when the auricular muscle was already extensively diseased. But Dr. Smith gave the impression of claiming that where cardiac failure occurred without a change of rhythm, it was very likely not attributable to exertion. When fibrillation of the auricles set in, it was due to a change in the fibres which lost, at least temporarily, much of their contractility. Lewis had proved that by his demonstration of "circus movement." However, between the perfectly healthy heart containing fibres incapable of being strained, and the diseased heart capable of fibrillating under the influence of exertion, there must be all grades of damage to which the muscle could be subjected by effort. It was possible for a man to jeopardize his chances of recovery by performing hard work when he had a diseased heart, even if fibrillation did not result from the effort. Dr. Smith had said that there was some doubt on this point. But if the muscular fibres could fail to the point of fibrillation as a result of exertion, then they could fail to a less extent when the normal rhythm was preserved. What more or less permanent changes occurred in the muscular fibres when the diseased heart was subjected to effort, no one knew. They had to assume that the contractility of the fibres was injured. Metabolites, such as carbon dioxide and lactic acid, could permanently injure the heart muscle, and it was possible that when the fibres had defective nutrition these poisons which accumulated with exertion, produced permanent harmful effects. The old custom of keeping patients suffering from cardiac failure continuously in bed for a period of six months or more had much to commend it. The prolonged rest and freedom from exertion led to a remarkable increase in the cardiac reserve, provided the disease process had not advanced to a condition beyond repair.

Dr. McDonald expressed his interest in Dr. Corlette's application of the principles of mechanics to many of the problems of trauma in visceral disease. He held the same opinions as Dr. Corlette in regard to perforation of a peptic ulcer. It was difficult to accept the view that a gradual rise in intraabdominal pressure could cause perforation. The chief factor in perforation was ulceration, and even when a chronic ulcer perforated, it did so because one portion was in an active ulcerating stage, gradually eating its way through the peritoneum. In ordinary circumstances the only influence hastening the perforation was a rise in intragastric pressure as, for example, after a copious drink or a full meal. Dr. Corlette's remarks on this condition had been sound and logical. There was only one point on which Dr. McDonald wished to comment: Although it was true that increased exertion caused an increase of intraabdominal pressure and that that pressure would not cause rupture of the ulcer because the pressure was exerted on all sides equally and because when the pressure outside the stomach was raised, the pressure inside was raised correspondingly—although this was true, yet Dr. McDonald could conceive of a violent jerk producing rupture of an ulcer which was on the point of bursting. There might not be sufficient time for the force to be distributed over the whole of the stomach. A violent punch causing rupture was in the same category.

DR. C. B. BLACKBURN said that he was very glad to have heard the two extremely interesting papers. He was, as a physician, more interested in that of Dr. Smith. He

entirely agreed with Dr. Smith in regard to heart failure and its inability to be induced by exertion in the healthy heart, and he also agreed with what had been said in regard to the damaged heart. But when a medical practitioner had to give evidence before a court, the court was incapable of appreciating medical standards of the failing heart. If they took, for instance, a man with a "toxic heart," perhaps from smoking too much, or as a result of some septic process, after some severe exertion he might for the first time manifest the effort syndrome; his heart would beat rapidly on slight effort; perhaps it had been suggested to him by his medical adviser that he had a "weak heart." In such conditions it was not always possible to persuade the patient or the court that it was not a case of "weak heart." The question would be asked: "Is this man as well able to do his work with his heart beating at 100 a minute?" Of course he was not. Until the doctor could stop this rapid beat, the problem remained. It was difficult to explain to a court, because obviously the patient was not the man that he had been before.

As regards coronary occlusion, he was doubtful as to whether in this condition there was always a thrombosis. Identical symptoms could be due to a hæmorrhage; the muscle would then be impregnated with blood at the point beyond where a coronary vessel was narrowed. This might be the true explanation of those cases presenting the symptoms of coronary occlusion after an effort. Dr. Blackburn recalled the case of a country man who collapsed when running after some sheep and was found to have coronary occlusion. This attack had undoubtedly been the result of extra effort. Similar attacks had been known to occur during a game of golf. He did not see why a clot should occur under those conditions, but it was possible that it might. Most cases unquestionably did occur when the patient was at rest, but they did occur at times after effort.

PROFESSOR C. G. LAMBIE congratulated the speakers and remarked upon the intricate problems raised; some of these were almost akin to that of the meeting of the irresistible force and the immovable object. The introduction of the formula $\frac{1}{2}mv^2$ had filled him with admiration. He congratulated Dr. Smith on the able way in which he had treated the matter and the clear exposition that he had given of the relationship between the physiology of circulation and the changes met with in disease. He wished to comment on one or two points. Dr. Smith scouted the question as to whether the heart dilated because the diseased muscular fibres became more easily stretched. They must turn to the question of "tone." There was doubt as to the existence of "tone" or what should be defined as "tone" in heart muscle. It was a bad expression, because two factors were concerned: (i) contractibility and (ii) distensibility of the cardiac muscle. How much stretching would occur in different hearts for the same amount of intracardiac pressure? When the ill-nourished heart was subjected to the same pressure as the normal heart, it distended more readily.

As regards the pericardium, they had to consider what happened in the normal heart and what happened in the diseased heart. The pericardium had been shown to be capable of resisting a considerable force in the healthy heart. In the diseased heart there were two factors to consider: If the force on the pericardium was exerted over a long period, then it became stretched. If the heart enlarged suddenly, then it must previously have been stretched, or else the pericardium must have been the seat of some disease that decreased its power to resist. It seemed that there was great room for experimental work in this quarter. Professor Lambie hoped that some of those present would undertake such work.

Professor Lambie then referred to the possible causes of dilatation of the heart without abnormaltiy of rhythm. He thought that exercise played a part in causing dilatation when the myocardium was dilated or poisoned, as when patients suffering from influenza were allowed to get up and go about their work too soon. These patients needed prolonged rest before the heart recovered. Why was it that some hearts fibrillated after exercise and others did not? When fibrillation of the auricles was brought about experimentally by electrical stimulation it lasted only for a short time; therefore it was doubtful whether it could be stated with certainty that, when fibrillation persisted, the heart muscle could be registered as having been absolutely healthy from the start.

PROFESSOR HAROLD DEW said that Dr. Corlette's paper was, as usual, suggestive and thoughtful, but he questioned, however, this particular application of pure physics to the abdomen. Only if the organs were made of inert material would the arguments advanced be valid under all conditions. When there was a rise of pressure in the abdomen, the stomach had to take up the pressure too, and once the pressure was relieved, the stomach had to relieve its pressure also. He thought that it was possible to get rupture from an ordinary strain, although all were agreed that the usual cause of rupture was a rise of intragastric pressure after ingestion of food or drink. Professor Dew quoted an instance of a man who had jumped over a low fence while returning home from work. He was seized with violent pain while in the act of jumping, collapsed and was operated on three hours later for perforated duodenal ulcer. The patient had had an ulcer on the point of rupture, and the stomach had not had time to take up the sudden increase of pressure. Undue resilience could not be expected of the organ, especially if part of its wall was weakened by disease.

Dr. Corlette, in reply to Dr. Storey's question as to what advice he would give to anyone in whom he had a special interest, if that person had an ulcer on the point of rupture, stated that if the question had been different, there would have been a different answer. If it were a question of a man either "starving or busting," would he advise that man to take food and drink? But if it were merely a question of walking up stairs . . . Anyhow, how did he know if the ulcer was on the point of rupture? As to Dr. Storey's experiences of torsion of the cord, the condition he saw may have been torsion of the cord; but if it was, then Dr. Storey's experience was a very unusual one; Dr. Corlette himself had never seen such a case. Perhaps they were all blind, or perhaps the others were too wide awake.

Dr. Corlette knew that it was possible for people who had varicocele to live not to have it; it disappeared. When injections were made into varicose veins, cure resulted with the formation of clots. That was what he had meant in speaking of the natural process of cure.

In reply to Dr. McDonald's suggestion that a sudden jerk might rupture an ulcer, Dr. Corlette conceded that it might under certain conditions—for instance, if the ulcer contained a considerable amount of fluid. That was where the formula $\frac{1}{2}mv^2$ came in. It was a question of mass, the mass of the stomach contents and the velocity of the jerk. Dr. Corlette believed that this physical formula held universally. He referred to the case cited by Professor Dew, where it would look to the plain man as though the rupture came because the patient was getting over the gate; but did the rupture occur during this operation or as a result of it? It was impossible, moreover, to judge by one case. If many cases were discovered, then he would begin to think that there was something wrong with his reasoning. All cases must be considered on their merits. It might be further remarked that if the stomach perforated through strain of an ulcer caused by a stress applied from without, the rupture would be a "blow-in" type, not a "blow-out." It would rupture from without inwards, not from within outwards. Professor Dew had given him a bit of a shock, however, What he said amounted to a suggestion that the physical formula $\frac{1}{2}mv^2$ was contrary to common sense and therefore could not be accepted in matters of the sort. Did it apply? All Dr. Corlette could say was that it did apply.

Dr. Smith, in reply to the questions raised during the discussion of the papers, said that he was going to pass by Dr. Storey's remarks. Dr. McDonald, he said, had merely put forward the vague conventional view of heart failure following exertion. Dr. McDonald, while admitting that effort might produce fibrillation in a diseased heart, appeared to take that to be evidence of over-distension of the heart muscle fibres. It was not so. A possible explanation of fibrillation after exercise was that, as the heart

rate rose in a heart ready to fibrillate, one stimulus to the auricular muscle followed another at just the appropriate interval to initiate a circus movement; then fibrillation occurred and failure eventuated because the ventricular contractions became unable to maintain the systolic output.

In regard to Dr. Blackburn's comments on coronary occlusion and the possibility of rupture of the coronary artery, Dr. Smith had not considered the question of rupture. He thought that when occlusion occurred in times of activity, it was not necessarily because of the effort, it was merely coincidental. But he could conceive in the coronary system of small plaques being driven on with increased coronary circulation and being followed by embolism. He had not thought of infarction of some of the areas being due to rupture and hæmorrhage. Dr. Smith agreed with Dr. Blackburn as to the difficulty of convincing the court that the patient had not a diseased heart if the heart rate was high. He also agreed with Dr. McDonald that if they could get to the patient early enough, the damage to the mind could be arrested. With patients in whom recovery occurred, Dr. Smith had noticed that if the opinion expressed by the medical practitioner was supported by the court, it had a potent influence on the patient.

In regard to Professor Lambie's mention of the "tone" of the heart muscle, he said that he had not read the whole of the paper that evening on account of its length. The question of tone was included in the parts omitted. Dr. Smith then read portion of the omitted sections of his paper.

## NOMINATIONS AND ELECTIONS.

THE undermentioned have been elected members of the Queensland Branch of the British Medical Association:

Williamson, William Stanley, M.B., B.S., 1928 (Univ. Sydney), Cox Street, Toowoomba.

Pittar, Cecil Arthur, M.B., B.S., 1930 (Univ. Sydney), Brisbane Hospital, Brisbane.

Guinane, James Vincent, M.B., B.S., 1927 (Univ. Sydney), Townsville.

# Obituary.

## ARTHUR EDWARD MORRIS.

DR. ARTHUR EDWARD MORRIS, whose death was announced in a previous issue of this journal, was born in November, 1879, at the Melbourne Grammar School, where his father, Professor Edward Ellis Morris, was headmaster. He was educated at Melbourne Grammar School and at Rugby, England. At Rugby he was in the first eleven; he was also in the football team, but it was as a cricketer that he made his name at school.

Returning to Australia, he entered Trinity College, University of Melbourne, as a medical student. He was successful in his studies. He gained first class honours in anatomy and first class honours and a scholarship in surgery. He graduated Bachelor of Medicine and Bachelor of Surgery in 1903. During his undergraduate days he gained his blue for cricket.

After graduation Morris served for a period as Resident Medical Officer at both the Melbourne Hospital and the Children's Hospital, Carlton. For some time he was Medical Tutor at Queen's College. He went abroad in 1908 and after doing special work in Vienna and Paris, he went to London, where he was appointed House Surgeon at Saint Peter's Hospital. In 1909 he came back to Australia and started practice as a genito-urinary specialist.

During the war Morris served first of all with the Royal Army Medical Corps, first at Langwarrin and later in France. In 1916 he was transferred to the Australian Army Medical Corps and was sent to Egypt, where he acted as adjutant to the first Australian Dermatological Hospital. He was with the same unit at Bulford Hill until July, 1919, when he returned to Australia. After the war Morris devoted much time and study to his specialty, and at the time of his death he was in charge of the Venereal Disease Clinic at the Melbourne Hospital.

Morris was a keen fisherman and bridge player—he was President of the Regent Club, a Melbourne bridge club. He was for many years honorary treasurer of the charity organization known as Morris House, which was founded by his father. He was a man with an understanding mind and wide sympathies.

# Correspondence.

## SNAKE-BITE.

SIR: In your issue of October 10 I note a letter by Dr. Jacobs in which he expresses his disbelief in the view that "should a snake-bitten person show signs of drowsiness, he must forcibly be kept awake." I am in full agreement with Dr. Jacobs. There can be no question that what a snake-bitten person needs is rest, both of body and mind, and no treatment could be worse than that often administered by ignorant persons, of walking him up and down, or even inflicting physical pain, with a view to combating the drowsiness which is an early symptom of bites by Australian snakes.

Yours, etc.,

CHARLES H. KELLAWAY.

Walter and Eliza Hall Institute,
Melbourne,
October 14, 1931.

## TONSILLITIS.

SIR: It was with satisfaction that I read the timely article on chronic tonsillitis coming as it does from a surgeon of accepted eminence in Dr. R. Graham Brown. Two features are particularly worthy of stress in these times, when quite unwittingly, I believe, patients (and their doctors) are deceived by the belief that adult diseased tonsils are adequately and completely removed either by guillotine or by diathermy. I would not condemn the guillotine in every adult case, especially if the tonsil is firm and mobile, while the operation is performed without haste and with good light and hæmostasis such that the surgeon finally sees a completely emptied tonsil fossa. That class of case, however, in which the pharyngeal branches of the lower pole are in evidence, cannot be completely dealt with by other methods than dissection, yet if incompletely handled, is liable rapidly to bring discredit, for these already diseased lymphoid extensions rapidly enlarge, if not to refill the tonsil fossa, then at least to give rise to recurrence of sore throats, laryngitis and cervical adenitis, to say nothing of persisting focal infection. I would, however, further stress a point of experience, that where the retrofaucial or infratonsillar lymphoid masses show disease, there is very often to be found either oral or nasal sepsis; such feeding the tonsil before operation now makes call upon the remaining lymphoid structures, causing their enlargement and often inflammation, a beacon light directing attention to the mouth and sinuses as well as calling for eradication of the diseased lymphoid remains.

Dr. Graham Brown's second timely comment is upon diathermy for removal of tonsils. In brief, can the surgeon claim ability to stay coagulation neither short of nor beyond that almost microscopical and invisible plane of areolar tissue referred to as the tonsil capsule? I have watched the whitening ring of coagulation form around the diathermy needle, mindful that its margin is beyond that visible to the naked eye, and that in the deeper unseen planes unexposed to air cooling it must be relatively more extensive still, yet one cannot see those depths; either tonsil tissue extends beyond the burn or muscle is included in the destruction. Complete tonsil destruction without muscle burning, I am convinced, is by this method

impossible, and its attempt unscientific. Further, I cross swords with those who claim tonsil sterilization. Such does not occur; remaining tonsil structure there will be, and such rapidly exhibits signs of infection again. In cases of focal reaction, often grave disease is liable to be temporized with. Further, the method is not always without pain nor without danger of hæmorrhage. There are rare cases unsuitable for operation, with recurring sore throats, which are relieved by diathermy. Extensively practised as it is today, and especially in endeavours to remove septic foci, the method should be condemned ere it brings further discredit upon our profession, too often accused of being unscientific.

Yours, etc.,

DOUGLAS G. CARRUTHERS, M.B., Ch.M.,
*Honorary Assistant Ear, Nose and Throat Surgeon, Sydney Hospital; Consulting Ear, Nose and Throat Surgeon, Canterbury Hospital.*

Sydney,
October 8, 1931.

## ANGINA PECTORIS AND CORONARY OCCLUSION.

SIR: Dr. Blackburn, in his article on "Angina Pectoris and Coronary Occlusion," suggests involvement of dorsal nerves in a spondylitic process as a probable cause of anginal attacks, and cites five illustrative cases.

The average age of these patients was fifty-four years. Radiological examination of any series of patients of this age suffering from any disease whatsoever will reveal a large number of unsuspected spondylitic changes. Comparatively few of these patients ever have any symptoms referable to spondylitis. It must be an exceedingly small proportion even of these few in whom such symptoms take the form of sudden attacks of intense pain.

The anginal attacks in the cases quoted by Dr. Blackburn were all classical as regards type of pain, distribution of pain and relationship to effort. In four of the five some pathological cardiovascular condition was demonstrable; the fifth was not beyond suspicion. One of these patients has already died a cardiac death.

It seems hardly necessary to invoke a mechanism whose very existence is not definitely established, to explain the occurrence of anginal pain in patients in whom there is more than reasonable ground for belief that the generally accepted causes for such pain were present and functioning.

Yours, etc.,

BRUCE HUNT.

26 St. George's Terrace,
Perth,
October 17, 1931.

## PUERPERAL SEPSIS.

SIR: The correspondence regarding puerperal sepsis and its treatment has suggested the advancement of the following simple prophylactic suggestion. Today medical science strives after the ideal of asepsis in that the use of antiseptics may, if over relied upon, tend to carelessness in asepsis. With the ever-present anus and its possibility as a source of infection, is asepsis always achievable in midwifery and is not antisepsis rather a justifiable procedure? After the procedure about to be described I have not seen puerperal fever. And as cass are included of contamination in dirty shroudings, such as holding the uterus for a quarter of an hour after a severe *post partum* hæmorrhage following manual removal of the placenta on dirty bed linen, design as well as good fortune may be involved. Briefly, it has been the writer's experience to find road wounds, even with tendons stripped of their coverings, heal by first intention when biniodide of mercury (solution one in a thousand) has been packed in the wound for five minutes before suturing. This result has been invariable.

In virtue of this, before manual interference with a parturient woman, the writer has squeezed the fluid from a large swab soaked in biniodide of mercury into the vagina. This can be repeated with each intrauterine interference. After delivery which has demanded any considerable need for introduction of the hand into the uterus, a large swab steeped in the solution is placed in the vagina on short forceps, so as to be against the os for an hour.

If this simple proeedure were adopted, puerperal sepsis may cease to exist owing to the unique bactericidal powers of biniodide and what appears an equally unique diffusibility.

Yours, etc.,

"PHYSICIAN."

Melbourne,
October 20, 1931.

# Proceedings of the Australian Medical Boards.

## TASMANIA.

THE undermentioned have been registered under the provisions of the *Medical Act*, 1918, of Tasmania, as duly qualified medical practitioners:

Robertson, William Lincoln, M.B., B.S., 1913 (Univ. Melbourne), Brighton, Victoria.
Phillips, Lewis Leon, M.B., B.S., 1927 (Univ. Melbourne), Malvern, Melbourne, Victoria.

# Congresses.

## CONGRESS OF THE PAN-PACIFIC SURGICAL ASSOCIATION.

SIR JAMES BARRETT has received from Dr. F. J. Pinkerton, Secretary of the Second Congress of the Pan-Pacific Surgical Association, a letter in which reference is made to altered arrangements for holding the congress. Sir James Barrett has been kind enough to forward the letter for publication; portion of it is reproduced herewith. Those who contemplate making the journey to Honolulu, will note with satisfaction that the congress is to be held in the cooler part of the year. Dr. Pinkerton writes as follows:

You have probably heard that the Second Congress of the Pan-Pacific Surgical Association is to be held the last week of April or the first week of May in 1933. This change was decided upon because of the Olympic Games in Los Angeles in 1932 and the world-wide depression which undoubtedly would interfere greatly with the number of delegates if the meeting were held in 1932. I have just addressed a letter to James Hardie Neil and informed him of this change in plans.

From the interest shown from various doctors on the mainland, and the number of inquiries we have had, lead us to believe that the second Congress will be more successful and more largely attended than the first, which is, of course, very gratifying. We are counting on a goodly number from your section of the Pacific, and any suggestions that you have to offer will be greatly appreciated.

Our local committees have, in the main, been appointed, and delegates can count on strong general support from our local physicians. The present consensus of opinion seems to be that the Congress will be composed chiefly of symposia on subjects of major importance, the whole surgical field being divided into specialties with chairmen of each group. The appointment of definite chairmen has not been entirely completed, but as soon as we are certain of the appointments, we will inform you. Because of the success of the round table breakfast conferences at the last Congress, and the increased popularity of the round table conferences, it has been definitely decided to extend the time to two hours for each breakfast conference, dividing them up in such a way that everyone can attend each if he so desires.

## Books Received.

MEDICAL JURISPRUDENCE, by C. Scheffel, Ph.B., M.D., LL.B.; 1931. Philadelphia: P. Blakiston's Son and Company. Demy 8vo., pp. 325. Price: $2.50 net.

MENTAL NURSING (SIMPLIFIED), by O. P. Napier Pearn, M.R.C.S., L.R.C.P., D.P.M.; 1931. London: Baillière, Tindall and Cox. Foolscap 8vo., pp. 312, with illustrations. Price: 5s. net.

HISTORY OF THE GREAT WAR BASED ON OFFICIAL DOCUMENTS: MEDICAL SERVICES, CASUALTIES AND MEDICAL STATISTICS OF THE GREAT WAR, by T. J. Mitchell, D.S.O., M.D., Ch.M., and G. M. Smith, M.B.E., M.A.; 1931. London: His Majesty's Stationery Office. Demy 8vo., pp. 402. Price: 21s. net.

GONORREA (BLENORRAGIA) IL MALE PIÙ DIFFUSO, DIFESA SOCIALE E PERSONALE, by F. Marta; 1932 (*sic*). Milan: Ulrico Hoepli. Foolscap 8vo., pp. 291, with illustrations.

LASSETER'S LAST RIDE, AN EPIC OF CENTRAL AUSTRALIAN GOLD DISCOVERY, by I. L. Idriess, with foreword by H. Basedow, M.A., M.D., B.Sc.; 1931. Australia: Angus and Robertson. Crown 8vo., pp. 241, with illustrations. Price: 6s. net.

## Diary for the Month.

Nov. 10.—New South Wales Branch, B.M.A.: Ethics Committee.
Nov. 11.—Victorian Branch, B.M.A.: Branch.
Nov. 12.—New South Wales Branch, B.M.A.: Clinical Meeting.
Nov. 12.—Victorian Branch, B.M.A.: Council.
Nov. 13.—Queensland Branch, B.M.A.: Council.
Nov. 17.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
Nov. 24.—New South Wales Branch, B.M.A.: Medical Politics Committee.
Nov. 25.—Victorian Branch, B.M.A.: Council.
Nov. 26.—South Australian Branch, B.M.A.: Branch.
Nov. 26.—New South Wales Branch, B.M.A.: Branch.
Nov. 27.—Queensland Branch, B.M.A.: Council.
Dec. 1.—New South Wales Branch, B.M.A.: Organization and Science Committee.
Dec. 1.—New South Wales Branch, B.M.A.: Ethics Committee.
Dec. 2.—Victorian Branch, B.M.A.: Council.
Dec. 2.—Victorian Branch, B.M.A.: Annual General Meeting.
Dec. 3.—South Australian Branch, B.M.A.: Council.

## Medical Appointments.

Dr. M. W. Phipps (B.M.A.) has been appointed Government Medical Officer at Barraba, New South Wales.

* * *

Dr. H. F. Hustler has been appointed a Resident Medical Officer at the "Mareeba" Babies' Hospital, South Australia.

* * *

Dr. J. Kenny (B.M.A.) has been appointed to act at Esperance, Western Australia, as Medical Inspector of Seamen, pursuant to the provisions of Section 123 of the *Navigation Act*, 1912-1926.

## Medical Appointments Vacant, etc.

**FOR announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser," page xvi.**

AUSTIN HOSPITAL FOR CHRONIC DISEASES, HEIDELBERG, VICTORIA: Honorary Pathologist, Honorary Anæsthetist.

INFECTIOUS DISEASES HOSPITAL, FAIRFIELD, VICTORIA: Consultant Surgeon.

LAUNCESTON PUBLIC HOSPITAL, TASMANIA: Medical Superintendent.

MANLY DISTRICT HOSPITAL, SYDNEY, NEW SOUTH WALES: Honorary Consultant Radiographer, Honorary Radiographer, Resident Medical Officer.

SYDNEY HOSPITAL, SYDNEY, NEW SOUTH WALES: Honorary Vacancies.

THE WOMEN'S HOSPITAL, SYDNEY, NEW SOUTH WALES: Resident Medical Officer.

## Medical Appointments: Important Notice.

**MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.**

| BRANCH. | APPOINTMENTS. |
|---|---|
| NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney. | Australian Natives' Association.<br>Ashfield and District United Friendly Societies' Dispensary.<br>Balmain United Friendly Societies' Dispensary.<br>Friendly Society Lodges at Casino.<br>Leichhardt and Petersham United Friendly Societies' Dispensary.<br>Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney.<br>North Sydney Friendly Societies' Dispensary Limited.<br>People's Prudential Assurance Company, Limited.<br>Phœnix Mutual Provident Society. |
| VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne. | All Institutes or Medical Dispensaries.<br>Australian Prudential Association, Proprietary, Limited.<br>Mutual National Provident Club.<br>National Provident Association.<br>Hospital or other appointments outside Victoria. |
| QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane. | Members desiring to accept appointment in ANY COUNTRY HOSPITAL, are advised to submit a copy of their agreement to the Council before signing, in their own interests.<br>Brisbane Associated Friendly Societies' Medical Institute.<br>Mount Isa Mines.<br>Toowoomba Associated Friendly Societies' Medical Institute. |
| SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide. | All Lodge Appointments in South Australia.<br>All Contract Practice Appointments in South Australia. |
| WESTERN AUSTRALIAN: Honorary Secretary, 65, Saint George's Terrace, Perth. | All Contract Practice Appointments in Western Australia. |
| NEW ZEALAND (Wellington Division): Honorary Secretary, Wellington. | Friendly Society Lodges, Wellington, New Zealand. |

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